Public Health

FEB 7 1961

FEBRUARY 1961

VOL. 3 NO. 2

Journal of Occupational Medicine

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1. Greene, C. C., Jr., and Beaver, G. T.: Journal of Occupational Medicine 2:*363, 1960. 2. Influenza Fact Sheet: Public Health Service, U. S. Dept. of Health, Education and Welfare, August 1, 1960. 3. Burney, L. E.: Correspondence,* J.A.M.A. 174:*438, 1960.*

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Official Publication of the Industrial Medical Association

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Journal of Occupational Medicine is published by the Industrial Medical Association, 28 East Jackson Boulevard, Chicago 4, on the 1st of each month. Second class postage paid at Chicago, Illinois. Copyright 1961 by the Industrial Medical Association. Title registered, United States Patent Office.

Subscriptions $10.00 per year; $25.00 for three years—U. S. and foreign. Single copies $1.00 postpaid. Make checks payable to Journal of Occupational Medicine.

Subscriptions, changes of address, and all business matters should be addressed to Clark D. Bridges, Publication Manager, Journal of Occupational Medicine, 28 East Jackson Boulevard, Chicago 4, Ill.

Manuscripts submitted for publication should be sent direct to the Editor, Adolph G. Kammer, M.D., 2057 Beechwood Boulevard, Pittsburgh 17, Pa.

Change of address notices received by the 15th of any month will assure delivery of the following month's issue. Address change is facilitated if old address is also furnished.

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Official Publication of the Industrial Medical Association

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SUGGESTIONS TO AUTHORS

Materials for publication should be addressed to the Editor, 2057 Beechwood Blvd., Pittsburgh 17. Articles will be considered for publication on condition that they are submitted solely to *Journal of Occupational Medicine*.

The manuscript, including references, footnotes, and legends for illustrations, should be typed on one side of a standard-sized page of opaque paper, double-spaced, and with at least a one inch margin at each edge. One carbon copy should accompany the original typescript, and the author should retain a carbon copy for use in reading proofs.

The authors' names should be accompanied by the highest academic or medical degrees that they hold; letters indicating membership in honor or professional societies will not be published. Authors who are in the armed services should not use civilian degrees in connection with their names. The city of each author should be supplied, and an address should be provided at the end of the manuscript for the use of readers who may wish to communicate with the senior author. If academic, hospital, or industrial connections are given for one author, they must be given for all other authors who have such connections. The authors are responsible for obtaining permission from the proper authorities to use the names of institutions.

References to the literature should be limited to those used in preparation of the manuscript; they should be typed on a special page at the end of the manuscript and should be in the style of those used in the scientific publications of the American Medical Association. Titles of all articles must be given.

Charts and drawings should be in black ink on hard, white paper. Lettering should be large enough to permit necessary reduction. Oversized illustrations should be photographed and a glossy print submitted. Prints of a bluish tinge will not reproduce well. Paper clips must not be used on prints, since the marks left will show in the reproduction. A label should be pasted to the back of each illustration showing its number, the author's name, an abbreviated title of the article, and an indication of the top of the picture. Each chart and illustration must have a descriptive legend, and each table must have a caption.

PRELIMINARY PROGRAM

46th Annual Meeting

APRIL 11, 12 AND 13—BILTMORE HOTEL, LOS ANGELES

INTENSIVE REFRESHER COURSES AND CLINICS IN TRAUMA

Special courses requiring advance reservation represent an innovation in the IMA program. Admission is by ticket only, no fee. Persons desiring to participate should indicate the session they wish to attend by letter of application to the IMA headquarters office, 28 East Jackson Blvd., Chicago 4, Ill. Requests will be acknowledged; however, the tickets will be held at the Conference Registration Desk to be picked up by 5:00 P.M. the day prior to the session. Tickets not called for at that time will be made available to others who wish to participate.

INTENSIVE REFRESHER COURSES

(Participation limited to 40, each course, each session)

Tuesday, April 11

A. CARDIOLOGY IN INDUSTRY
Faculty: *Richard Call, M.D.*
Course Coordinator: *Hallet A. Lewis, M.D.*
First Session: 8:30-10:30 A.M.
Repeat Session: 10:40 A.M.-12:40 P.M.

B. TREATMENT OF RADIATION INJURY
Faculty: *Gould A. Andrews, Jr., M.D.*
Harry Foreman, M.D.
W. D. Norwood, M.D.
George L. Voelz, M.D.
Course Coordinator: *Thomas L. Shipman, M.D.*
First Session: 8:30-10:30 A.M.
Repeat Session: 10:40 A.M.-12:40 P.M.

1. Medical Supervision of Workers Potentially Exposed to Radiation
   Lecturer: *Dr. Voelz*
2. Treatment of Individuals Who Have Excessive Deposition of Radioisotopes
   Lecturer: *Dr. Norwood*
3. Treatment of Skin Wounds Contaminated by Radioactive Materials
   Treatment of Radiodermatitis (Beta Ray Burns)
   Lecturer: *Dr. Foreman*
4. Treatment of Patients Suffering from Acute Radiation Injury
   Lecturer: *Dr. Andrews*

Wednesday, April 12

C. DERMATOLOGY IN INDUSTRY
Faculty: *Raymond R. Suskind, M.D.*
Course Coordinator: *Merle Bundy, M.D.*
First Session: 8:30-10:30 A.M.
Repeat Session: 10:40 A.M.-12:40 P.M.

Wednesday, April 12—continued

D. HEARING CONSERVATION IN INDUSTRY
Faculty: *Aram Glorig, Jr., M.D.*
Course Coordinator: *James P. Hughes, M.D.*
First Session: 8:30-10:30 A.M.
Repeat Session: 10:40 A.M.-12:40 P.M.

CLINICS IN TRAUMA

(Participation limited to 50, each clinic)

Each subject will have an instructor highly qualified in the particular field of discussion. Each clinic will open with a 20 to 30 minute discussion of the general problem, to be followed by audience participation with participants presenting some problem cases of their own and general comments regarding their treatment. Those planning to attend should bring x-rays of a problem case to the meeting.

Tuesday, April 11

8:30-10:00 A.M.

E. FRACTURES AND CRUSH INJURIES OF THE FOREARM AND HAND
Herbert Kirchner, M.D., Presiding
Lecturer and Discussion Leader: *James N. Wilson, M.D.*

F. PROBLEMS OF THE BACK
Leon O. Desimone, M.D., Presiding
Lecturer and Discussion Leader: *Homer C. Pheasant, M.D.*

G. INJURIES OF THE LEG AND ANKLE
Packard Thurber, Jr., M.D., Presiding
Lecturer and Discussion Leader: *John C. Wilson, M.D.*

GENERAL PROGRAM

Tuesday, April 11

10:30 A.M.-12:00—General Session

REHABILITATION OF THE INDUSTRIALLY INJURED
—Panel Discussion with Case Presentations
Moderator: *Asa Barnes, M.D.*

INJURIES OF THE SPINAL CORD
Thomas Cucker III, M.D.

AMPUTATIONS
Charles O. Bechtol, M.D.

UPPER EXTREMITY FUNCTION IN PATIENTS WITH SEVERE CERVICAL SPINE INJURY
Vernon L. Nickel, M.D.

Association Activities

Tuesday, April 11—continued

INJURIES OF THE BACK
John H. Aldes, M.D.

2:00 P.M.—General Session
ADDRESS OF WELCOME
Robert E. Eckardt, M.D., President, Industrial Medical Association

2:10 P.M.—C. O. SAPPINGTON MEMORIAL LECTURE—sponsored by the Occupational Health Institute
Rutherford T. Johnstone. M.D.

3:00 P.M.—Intermission—Visit Exhibits

3:30 P.M.—Joint Session—Industrial Medical Association and American Association of Industrial Nurses
Margaret L. Steele, R.N., Presiding
President, A.A.I.N.
THE CONTEMPORARY SCIENTIST-ENGINEER
HIS EMOTIONAL CONFIGURATION
Speaker to be announced
HIS ADJUSTMENT TO THE INDUSTRIAL SETTING
John Weir, Ph.D.

Wednesday, April 12

8:30-10:00 A.M.—General Session—Medicine
THE CHEST AND INDUSTRY
Rutherford T. Johnstone, M.D., Presiding
PNEUMOCONIOSIS AMONG PENNSYLVANIA BITUMINOUS COAL MINERS
Jan Lieben, M.D., and *W. W. McBride, M.D.*
CANCER OF THE LUNG IN A PUBLIC UTILITY — A Fifteen-Year Study—1946-1960
Henry A. Christian, M.D.

10:00 A.M.—Intermission—Visit Exhibits

10:30 A.M.-12:00—General Session
RESUSCITATION TECHNIQUES
Ralph de Forest, M.D., Presiding
MOUTH-TO-MOUTH RESUSCITATION, WITH AND WITHOUT ADJUNCTS
Archer S. Gordon, M.D.
THE RATIONALE FOR THE CHANGE IN RESUSCITATION TECHNIQUES IN INDUSTRY
Roger C. Plaisted
RESUSCITATION TRAINING AND APPLICATION OF TECHNIQUES IN INDUSTRY
Jack Story

2:00 P.M.—ANNUAL BUSINESS MEETING
Presentation of Awards
Reports of Officers
Election of Officers and Directors

7:30 P.M.—IMA BANQUET AND DINNER DANCE
(No speeches)

Thursday, April 13

8:30-10:00 A.M.—General Session—Medicine and Surgery
ACHING FEET IN INDUSTRY
O. Tod Mallery, Jr., M.D., Presiding

Thursday, April 13—continued

THE CONTEMPORARY FIELD OF PODIATRY
Rayfield Aronow, D.S.C.
PODIATRY PROGRAMS IN INDUSTRY
Lawrence Hendel, D.S.C.

10:00 A.M.—Intermission—Visit Exhibits
10:30 A.M.-12:00—MENTAL HEALTH IN INDUSTRY—An Open End Discussion
Allan J. Fleming, M.D., Presiding
Moderator: *John H. Beatty, M.D.*
Participants: *Alec Bavelas, Ph.D.*
J. S. Felton, M.D.
Marvin Klemes, M.D.
Kenneth J. Munden, M.D.
The Rev. Clifford Peace

8:30 A.M.-12:00—General Session—Toxicology and Industrial Hygiene (Arranged by the Committee on Clinical Toxicology, IMA, and the Southern California Section, American Industrial Hygiene Association.)
INDUSTRIAL HYGIENE PROBLEMS IN THE MISSILE INDUSTRIES
Edward K. Daniels, Presiding
Speakers to be announced

10:00 A.M.-12:00—General Session—Surgery
John H. Leimbach, M.D., Presiding
ICE WATER TREATMENT OF BURNS
Alex G. Shulman, M.D.
NEW AND COMMON USES OF FELT FOR INJURIES
Norman W. Paul, M.D.
DIAGNOSIS AND MANAGEMENT OF BLUNT TRAUMA TO THE ABDOMEN
Gordon Smith, M.D.
LUMBAR SPINE DEGENERATIVE JOINT DISEASE—TO FUSE OR NOT TO FUSE
Homer C. Pheasant, M.D.
GYNECOLOGICAL PROBLEMS IN INDUSTRY
Robert J. McNeil, M.D.

1:30 P.M.—General Session—Medicine
Seward E. Miller, M.D., Presiding
A WORK CLASSIFICATION UNIT IN CARDIOLOGY
Robert Stivelman, M.D.
THE ENTRY OF ELECTRONIC COMPUTERS INTO MODERN MEDICAL PRACTICE
Charles Schoettlin, M.D.
FORMATS FOR THE FUTURE—ARE IMA MEETINGS IN NEED OF CHANGE?
Hallett A. Lewis, M.D.
PROJECT HOPE—THE STORY OF THE SS HOPE I, AND WORKERS ABROAD
William B. Walsh, M.D.

1:30 P.M.—General Session—Surgery
Leonard Arling, M.D., Presiding
TREATMENT OF THE PATIENT WITH MULTIPLE INJURIES—A Discussion Panel
Moderator: *D. John Lauer, M.D.*
Participants: *Robert C. Combs, M.D.*
J. J. Crane, M.D.
John C. Jones, M.D.
Theodore Kurze, M.D.
Francis McKeever, M.D.
Willard J. Zinn, M.D.

Journal of Occupational Medicine

In recognition of February as Heart month, this issue is devoted largely to relationships between physical effort and cardiac problems. Harry E. Ungerleider, M.D., New York City, served as guest editor for this issue. A final article in the project, by Richard S. Gubner, M.D., will be published next month.—Editor

HEART DISEASE AND EMPLOYMENT

Cardiovascular Studies on Lumberjacks

M. J. Karvonen, M.D., Ph.D., Helsinki, Finland; P. M. Rautaharju, M.D., Minneapolis, Minn.; E. Orma, M.D., S. Punsar, M.D., J. Takkunen, M.D., Helsinki, Finland

WHEN THE human organism is examined as a machine, the same concept of wear and tear often tends to be applied to it as to inanimate machines. The more intense the use of the organism, the faster will it wear out. In the field of cardiovascular disease, this concept would make avoidance of exercise a potent means of prevention and cure. Such reasoning has, in fact, found an expression in the advice given in the past to many weakly children and patients suffering, or supposed to be suffering, from various forms of cardiovascular disease.

The present era with its impetus on rehabilitation has seen old beliefs shaken also in this field. In comparing various occupational groups, high frequency of atherosclerotic heart disease has been associated with sedentary or semisedentary life and exercise has been ascribed a protective action. However, such occupational differences have not been demonstrable in all studies. Even where exercise appears to protect against the clinical manifestations of atherosclerosis, the mechanism of an eventual protection is open to various interpretations.[1, 2]

Clearly, more studies are needed on this subject. An obvious approach is to find groups at the extreme ends of the exercise scale, people entirely sedentary and, on the other hand, physically very active. The work of the lumberjack is known to require more energy than any other occupation. Recorded energy expenditure figures range from 4,500 to 8,000 kilocalories per day.[3] If physical work affects the cardiovascular system in any occupation, then evidence for such an effect should be found among the lumberjacks.

A clinical and electrocardiographic study was made of 815 men in the county of North Karelia, East Finland, in the fall of 1959. Men from 40 to 59 years old were studied. All men in this age range living in a geographically defined region were invited to be examined; the response was as good as 98.4%. The main occupations in the area are forestry and small-scale farming; there is hardly any industry. Of the 815 men, 380 were engaged either fully or part-time in woodcutting.

Electrocardiography

The most objective tool for the study of cardiovascular disease among the living is elec-

From the Institute of Occupational Health, Helsinki, Finland, and the Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minn.

Support for this study has been received from the National Heart Institute, National Institutes of Health, Bethesda, Md. (Grant No. H-92-60); from a grant from Yrjö Jahnsson Foundation to the Finnish Heart Association; and from Sigrid Juselius Foundation, Finland.

Dr. Karvonen is Director of the Physiological Department, Institute of Occupational Health. Dr. Rautaharju is a Research Fellow of the Minnesota Heart Association. Drs. Orma, Punsar, and Takkunen are Research Fellows of the Finnish Heart Association.

Submitted for publication Dec. 3, 1960.

trocardiography. In epidemiological studies, the techniques and criteria of electrocardiography must be strictly standardized. In examining an individual patient, the clinical significance of each electrocardiographic anomaly has to be weighed in the light of all other evidence; in an epidemiological investigation, however, exact results are best available through the systematic recording of electrocardiographic changes, without an attempt to interpret their eventual significance in each individual case.

ECG criteria and a classification scheme have been developed and tested by the Laboratory of Physiological Hygiene, University of Minnesota.[4] The 12-lead electrocardiograms of the lumberjacks were compared with those of all men in other occupations in the same area. The results of the resting electrocardiograms are shown in Table I. A number of significant differences were observed. Q and QRS changes were approximately five times as common among the men in other occupations as among the lumberjacks. Although the absolute numbers are small, the difference is clearly significant ($P = 0.01$). ST changes which may be ascribed to myocardial ischemia were similarly approximately four times as common in the lighter occupations; with the T wave items the difference was less marked, the ratio being less than 2 to 1. A closer analysis of the electrocardiograms reveals that each degree of Q and QRS changes was commoner among the men in lighter occupations than among the lumberjacks. The same applies to different degrees of ST and T changes.

No differences were observed in the anomalies of atrioventricular or ventricular conduction, in arrhythmias excluding sinus tachycardia and bradycardia, and in the group of miscellaneous electrocardiographic changes. The last group is relatively large: The main contributions come from "qualitative" T-wave findings including high or peaked T, postextrasystolic T-wave inversion, T notching, etc. (total 78 cases); from QRS abnormalities such as notching, slurring, RR prime, etc. (total 57 cases); and from low QRS amplitude (in leads I, II, III no positive or negative deflection greater than 5 mm or maximum QRS amplitude less than 10 mm in leads V_1-V_6; (total 22 cases). No significant difference was observed in any of these subgroups between the lumberjacks and the men in other occupations.

With one exception, the QRS axis deviations were all left axis deviations. Even in this group a majority of the changes belonged to the lighter occupations.

High amplitude R waves and sinus bradycardia are the only items which were commoner in the lumberjacks than in persons in other occupations. (With sinus bradycardia, the difference does not attain statistical significance.) These changes are directly associated with physical training. Long-distance runners and skiers are known to have a slow resting pulse rate.[5] High amplitude R waves are similarly a characteristic of endurance athletes.[6] The work of a lumberjack clearly has an element of endurance training.

Table I—ECG Findings at Rest
12-lead electrocardiograms
Rural men aged 40-59, East Finland, 1959
Absolute numbers in brackets

Category	Lumberjacks	Other Occupations	Total	*P*
Total Electrocardiograms	380	435	815	....
	Findings per 100 electrocardiograms			
I Q and QRS patterns	0.8 (3)	3.9 (17)	2.5	0.01
II QRS axis deviation	1.3 (5)	4.1 (18)	2.8	0.05
III High amplitude R waves	20.5 (78)	12.2 (53)	16.1	0.01
IV ST junction and segment	1.6 (6)	6.2 (27)	4.0	0.01
V T-wave items	8.4 (32)	13.1 (57)	10.9	0.05
VI A-V conduction	2.9 (11)	3.0 (13)	2.9	....
VII Ventricular conduction	3.2 (12)	2.8 (12)	2.9	....
VIII 1-6 Arrhythmias, except sinus tachycardia and bradycardia	3.2 (12)	3.0 (13)	3.1	....
VIII 7 Sinus tachycardia	1.3 (5)	2.3 (10)	1.8	....
8 Sinus bradycardia	3.7 (14)	1.6 (7)	2.6	....
IX Miscellaneous	21.2 (82)	22.3 (97)	22.0	....

An exercise test was made on all subjects for whom it was feasible and considered free from danger. The exercise consisted of stepping on a bench 30 cm high during three minutes at the rate of 20 steps per minute.

The exercise test was omitted for 5 lumberjacks (in one of these for technical reasons) and for 51 men of the other occupations. Moreover, the test was interrupted in 10 lumberjacks and in 19 men in the other group.

Immediately after the exercise test a new electrocardiographic examination was made. The test provoked 6.2 significant changes per 100 electrocardiograms (Table II). These were rather evenly distributed between the two occupational groups. There was some tendency toward more frequent occurrence of new ST changes among the lighter occupations, but

Table II—ECG Findings After Exercise
12-lead electrocardiograms
Rural men aged 40-59, East Finland, 1959
Absolute numbers in brackets

Category	Lumberjacks	Other Occupations	Total	*P*
Total electrocardiograms	375	384	759	....
	Findings per 100 electrocardiograms			
XI ST items *only* after exercise	2.7 (10)	5.8 (22)	4.2	....
XII T items *only* after exercise	0.0 (0)	0.3 (1)	0.0	....
XV Arrhythmias *only* after exercise	1.9 (7)	0.5 (2)	1.2	....
XVI Miscellaneous changes after exercise	0.8 (3)	0.5 (2)	0.7	....
Total	5.3 (20)	7.0 (27)	6.2	....
Exercise test stopped	2.7 (10)	4.9 (19)	3.8	....

owing to the small number of cases, the difference did not attain statistical significance.

The postexercise electrocardiogram thus brought out no differences between the occupational groups, in contrast to the marked differences observed in the resting electrocardiograms.

In international mortality statistics, Finland stands out in Europe as having the highest mortality from atherosclerotic heart disease (International List Nos. 420-422). The eastern part of Finland appears to be particularly afflicted. The high frequency of Q and QRS changes ascribable to past infarction falls in line with the mortality statistics. It is interesting to note that the main share of the ECG pathology belongs to men in the lighter occupations. In fact, the frequency of Q and QRS changes for the other occupations than lumberjacks, 3.9 per cent, is higher than that recorded in any of the populations of the same age studied by Keys and his co-workers (personal communication), including United States railroad personnel.

Selection or Protection by Exercise?

The present results directly indicate that atherosclerotic heart disease is less common in men engaged in heavy occupational work than among other occupations.

In laboratory studies on experimental animals, the concept of an adequate control group has been firmly established. It would appear desirable to apply the same principle also to human studies. In short-term experiments, this may easily be done; but in long-term research, the observance of the principle becomes difficult. When the period of observation extends over years, some subjects are bound to leave the experimental group. Among the lumberjacks, there certainly operates continuous selection; only the fit can stay on in the occupation. The "other occupations" of today consist to some extent of lumberjacks of the past. Obviously, one is not likely to meet men severely ill or invalid as active lumberjacks.

The self-selection which leads to the primary formation and further shifting of occupational groups causes a complete failure of the concept of the control group.[8] There is just as little possibility of choosing an ideal control group for the Karelian lumberjacks as, e.g., for the New York taxi drivers. The nearest that can be achieved is to choose a group of men who are not lumberjacks but geographically and socially as close to them as possible. Even so, it may always be argued that a difference between two occupational groups is due to spontaneous selection, social differences of various kind, etc., instead of—or in addition to—the factor to which attention is focused, in this case exercise. Where the experimental method is not applicable, there is no ideal answer; weighing the various possibilities remains always a matter of individual judgment.

The present results may give added proof to the view of Morris and co-workers[9] who see in exercise a factor protecting from the clinical manifestations of atherosclerotic heart disease. Their best-known evidence is provided by the London double-decker buses: The mobile conductors suffer less from this disease than the sedentary drivers. A point of interest in the present results was that a difference was manifest in all electrocardiographic items that are clearly consistent with atherosclerotic heart disease, but in no other items—e.g., in the categories of nonspecific abnormalities and arrhythmias. It is difficult to believe that selection would have excluded atherosclerotic heart disease from the group of lumberjacks, without having affected the frequency of other electrocardiographic abnormalities. The evidence thus is in favor of a specific prevention of atherosclerotic heart disease by heavy work. However, this conclusion is rather indirect and must be considered as tentative only. There is also the possibility that prolonged heavy occupational work may *produce* certain electrocardiographic abnormalities as indicated—e.g., by the significant difference in high amplitude R waves.

Other Etiological Factors?

In the well-known longitudinal study at Framingham, Mass., the individual risk of the

Table III—Serum Cholesterol
Rural men aged 40-59, East Finland, 1959
Mean ± standard error of the mean

	Lumberjacks	Other Occupations
Number	372	425
Serum cholesterol, mg per 100 ml	267.2 ± 1.75	265.5 ± 2.88

clinical manifestations of atherosclerotic heart disease has been shown to increase with high serum cholesterol, with high arterial pressure, and with cigarette smoking.[10-12] All these were included in the present study, in order to find out whether the occupational differences in electrocardiographic pathology may be due to differences in these factors.

Serum Cholesterol—In population comparisons, the serum cholesterol level has been found roughly to parallel the frequency of atherosclerotic disease. In fact, no instance of a population with high serum cholesterol and low atherosclerotic mortality or morbidity has been recorded, and the same applies vice versa.

The mean serum cholesterol level of the lumberjacks is compared with that of the other occupations in Table III. The values are rather high, as known from previous studies made in Finland.[7] However, there is practically no occupational difference.

In the Framingham study men with a serum cholesterol level exceeding 260 mg % had a three times higher risk of developing clinical manifestations of atherosclerotic heart disease than those below this limit. Within the period of six years, the risk was 1 in 11 for men aged 35-44 years, 1 in 8 for those aged 45-54 years, and 1 in 4 for the age range 55-62 years.[11] In the present study, more than half of the men have a "high-risk" cholesterol; a high frequency of past infarction fits well with the cholesterol situation.

Within some populations, those engaged in heavy manual work on an average have a lower serum cholesterol level than do people doing sedentary work. In other populations, the difference is not very marked. In a previous study in Karelia, the lumberjacks and farmers examined had approximately the same serum cholesterol level as men doing light work, over the age ranges 20-40 and 50-60 years. However, between 40 and 50 the cholesterol levels of the men doing heavy work were lower.[7] In the present total population study, no corresponding difference in this age range was observed.

In several instances, the lower serum cholesterol of men doing manual work has been associated with a diet containing relatively less fat than that of corresponding sedentary or semisedentary groups.[1] However, exercise has also been shown to prevent a rise of serum cholesterol as caused by a diet rich in saturated fatty acids.[13, 14]

In the present study, no difference was observed in the cholesterol level, but nevertheless the frequency of electrocardiographic evidence of atherosclerotic heart disease was widely different in the two groups. This is a notable result, and—probably for the first time—definitely indicates that the frequency of atherosclerotic heart disease in a population may be determined by factors other than the mean serum cholesterol level.

If the lumberjacks are protected from atherosclerotic heart disease by exercise, the protection is not mediated by a lower serum cholesterol level. Accordingly, the present results may be interpreted rather as demonstrating a "protective" action of exercise, independent of the serum cholesterol level. Also this conclusion is, of course, tentative only; the differences may still be due to selection, as discussed above.

Arterial Pressure—It is well known that systolic pressure rises and diastolic pressure falls during strenuous physical work. In trained athletes, the average arterial pressure at rest may be slightly lower than in the general population.[15]

In Table IV, the arterial pressure of the lumberjacks is compared with that of the men in other occupations. Both the systolic and the diastolic pressure are slightly lower in the lumberjacks. The lumberjacks had lower systolic and diastolic pressures than men in other occupations; hence, the lower arterial pressure might explain the lower frequency of electrocardiographic changes. However, the difference in pressure was rather small. It has also to be taken into account that the decisive factor may not be the resting, but rather the habitual arterial pressure. In strenuous exercise, the systolic pressure is known to rise considerably; thus, the lumberjacks may, nevertheless, have a higher 24-hour average blood pressure than the men in other occupations. The lower frequency of electrocardiographic changes among the lumberjacks therefore may hardly be

Table IV—Arterial Pressure
Rural men aged 40-59, East Finland, 1959
Mean ± standard error of the mean

	Lumberjacks	Other Occupations
Number	369	421
Systolic pressure, mm Hg	147.3 ± 0.99	149.2 ± 1.06
Diastolic pressure, mm Hg	89.5 ± 0.51	90.5 ± 0.68

Table V—Smoking Habits
Rural men aged 40-59, East Finland, 1959
Absolute numbers in brackets

Smoking Cigarettes	Lumberjacks n = 371	Other Occupations n = 46	Total n = 817	*P*
Never	8.4 (31)	17.3 (77)	13.2	0.001
Stopped	19.4 (72)	17.3 (77)	18.2	..
Less than 10 per day	8.9 (33)	11.4 (51)	10.3	..
11-20 per day	27.8 (103)	26.0 (116)	26.8	..
More than 20 per day	35.3 (132)	29.6 (125)	31.5	0.05

ascribed to a protective effect of low arterial pressure.

Smoking—In extensive statistical studies, smoking, particularly cigarette smoking, has been shown to increase the risk of cardiovascular disease.[16] In Finland, pipe and cigar smoking is very uncommon, and cigarettes are the main form of tobacco used.

A smoking questionnaire was presented to the men. A summary of the results is shown in Table V. The smoking habits of the lumberjacks do not differ much from those of the total population. If anything, the lumberjacks tend to smoke more; the percentage of nonsmokers was lower and that of heavy smokers higher than those among other occupations.

The smoking habits do not explain the differences in the frequency of ECG changes. If any difference should be expected as caused by smoking, it would be in the opposite direction than that observed.

Conclusions

Lumberjacks are men engaged in the physically heaviest occupation known. They show significantly fewer electrocardiographic changes indicative of past coronary infarction and of myocardial ischemia than all other men of the same age in the same area. This occupational difference is not adequately explained by differences in serum cholesterol, arterial pressure, or smoking habits. Selection and an eventual protective action of exercise, independent of the serum cholesterol level, remain as possible causes for the difference. A protection from artherosclerotic heart disease is suggested by the finding that in electrocardiographic items not specifically associated with atherosclerosis, like arrhythmias, there was no occupational difference.

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Occupational Physical Exertion and Coronary Atherosclerotic Heart Disease

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CURRENTLY, considerable confusion exists concerning the relationship of physical activity to coronary atherosclerotic heart disease. Furthermore, in those areas where well-documented information is available, new problems, rather than solutions, have been created. One reason for this unsatisfactory state of our understanding of the relationship of physical activity to coronary atherosclerotic heart disease is based upon the lack of proper definition as to which stage of the disease is under discussion in any particular investigation. For instance, the question as to whether the developmental stage of coronary atherosclerosis (the underlying chronic disease process) is being dealt with or whether the precipitation of acute events, such as sudden death, angina, or myocardial infarction in individuals who already have advanced coronary atherosclerosis, is under consideration is not usually clearly separated. These are obviously two different phases of the problem and wherever possible should be so defined. Also basic to the confusion is the fact that no completely satisfactory and proved hypothesis for the development of atherosclerosis exists. It has also been demonstrated that physical activity can affect factors in more than one hypothesis. For example, stressful physical activity affects not only serum lipid patterns but mechanisms concerned with the coagulability of blood.

Contributing to the difficulties are the serious inadequacies that exist in our methods of estimating physical activity and in our methods of selecting proper control groups. The acceptance of the realization that multiple causative factors are undoubtedly involved in the development of coronary atherosclerotic heart disease enhances the problem regarding proper control and interpretation of any study concerned with one factor, as, for example, in this instance occupational physical activity. There also exists the inability to separate the effects of psychological "stress" from physical "stress" as it pertains to any specific job activity. It has been recently demonstrated that the alterations in circulatory hemodynamics are quite similar regardless of the emotional or physical origin of the stimulus.

The purpose of this paper is to report some brief observations on two aspects of the problem, i.e., the relationship of occupational physical activity to the development of coronary atherosclerosis, and the effects of such activity, as well as unusual physical exertion on the precipitation of acute myocardial ischemic events in individuals with advanced coronary atherosclerosis. The findings in this report are based upon interpretation of the pertinent literature, as well as the author's personal postmortem observations and clinical studies on a population group of similar ethnic origins.

Occupational Physical Activity and the Development of Coronary Atherosclerosis

One of the earliest of the significant reports concerning occupational physical activity was that of Morris[1] and his associates in which they demonstrated that an association exists between the degree of physical activity in a particular occupation and the incidence of clinical coronary atherosclerotic heart disease. This study was conducted among employees of the transport system in London, in whom it was noted that the sedentary bus drivers manifested a significantly higher attack rate of angina and myocardial infarction than the more physically active ticket collectors. Other studies since then have, in general, arrived at similar conclusions, namely, that men in more physically active jobs have a lower incidence of coronary atherosclerotic heart disease,

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Some of the studies in this report have been supported in part by a grant from the Westchester Heart Association and by Grant No. H-2692 (C-1), National Heart Institute, U. S. Public Health Service.

Submitted for publication Dec. 1, 1960.

Table I—Distribution of White Men Who Died Suddenly from Acute Coronary Occlusion According to Occupational Physical Activity and Age

Age	Sedentary Activity		Moderate Activity		Strenuous Activity	
Years	No.	%	No.	%	No.	%
Under 55	112	44	81	31	35	24
Over 55	139	56	179	69	106	76
Total	251	100	260	100	141	100

especially under the age of 55, than those individuals of similar age in more sedentary occupations. It must be noted, however, that other studies have failed to confirm these findings fully. Nevertheless, a more recent study on death rates from heart disease among physically active and sedentary employees in the railroad industry concludes that the findings are consistent with the hypothesis that men in sedentary occupations have more coronary atherosclerotic heart disease than those in occupations which require moderate to heavy physical activity.[2]

In our postmortem studies of sudden death from coronary occlusion in males that have been conducted over the past 10 years in Westchester County, New York, it was noted that the men who had engaged in sedentary occupations tended to die suddenly from coronary occlusion at a younger age than those whose occupations involved considerable physical activity (Table I).[3] In this study, such occupations as bank executive, clerk, accountant, taxi driver were regarded as sedentary; such occupations as grocery clerk, letter carrier, were regarded as moderately active, and such occupations as road construction worker, plumber, steamfitter, were regarded as examples of strenuous activity.

Assuming that the clinical postmortem and epidemiologic evidence at present is sufficient to state that an association exists between the degree of occupational physical activity and the incidence of coronary atherosclerotic heart disease, the question arises as to possible mechanisms involved and the stage of the disease affected. Is the actual process of atherogenesis directly affected by the degree of lifetime physical activity, or are the morbidity or mortality rates from coronary atherosclerotic heart disease affected through some other relationship with occupational physical activity? In our own studies in which the degree of atherosclerosis in the coronary arteries on postmortem examination was measured in individuals who had died from various forms of accidents, no differences in the degree of atherosclerosis[4] were noted among the various occupational categories at similar age levels. The previously mentioned observations may, therefore, require a different explanation than the possibility that activity directly affects the atherogenic process. Another approach to the problem is indicated by Eckstein's study on dogs,[5] in which the experimental production of partial coronary occlusion was accompanied by the development of greater amounts of intercoronary anastomoses in the presence of exercise. The anastomotic channels that developed in these dogs were anatomically similar to those observed in human hearts. This would suggest that physical activity or the lack of it exerts an influence in the morbidity and mortality rates of clinical coronary atherosclerotic heart disease, not because of any direct effect on the atherosclerotic process, but perhaps indirectly through its stimulus to the development of collateral anastomoses. Thus, one might speculate that, given two individuals with the same degree of coronary atherosclerosis, the one whose myocardium is protected by more and larger anastomotic channels should be better able to cope with an emergency situation.

Currently, we are engaged in a study aimed at possible clarification of this particular point. In this study, all white males between the ages of 35 and 55 in a given community who have died as a result of industrial or automobile accidents are subjected to postmortem examination. The hearts are injected with plastic beads more than 40 μ (microns) in diameter into the main left coronary artery, and the rate of recovery from the right coronary ostea is noted. The degree of coronary atherosclerosis in the major coronary circulation is then measured. The information on the ability of particles larger than 40 μ to cross over from the left coronary vessels to the right coronary vessels is then correlated with the degree of coronary atherosclerosis and the lifetime occupational physical activity of the deceased subjects. As yet, the numbers of such cases so studied are insufficient to arrive at any conclusion.

Further evidence bearing on the relationship of occupational physical activity to the development of clinical evidence of coronary atherosclerotic heart disease is derived from our current study on white males of similar ethnic origins and cultural and geographic backgrounds, who have been somatotyped and are controlled as to blood pressure, weight, age, and occupational physical activity. In 3,000 Jewish white males now studied with standardized techniques, it was noted that 1 of every 50

individuals between the ages of 35 and 55, engaged in heavy physical activity had clinical evidence of coronary atherosclerotic heart disease, whereas 1 out of 33 individuals engaged in sedentary or light physical activity[6] had similar findings. The problems relative to the interpretation of these data as well as those reported by others, must include an understanding of the relationship of body form and diet to occupational physical activity. Proper interpretation also demands that we recognize that only little or no information exists on the accurate evaluation of the leisure-time physical activity of these individuals.

Occupational Physical Activity, Sudden Excessive Physical Exertion, and Precipitation of Acute Myocardial Ischemia

At any point in the course of advanced coronary atherosclerosis, acute episodes may occur. They vary in severity, duration, and effect on cardiac function, and are often unpredictable. In order of increasing significance, they are angina pectoris, acute coronary insufficiency, acute coronary occlusion with myocardial infarction, and/or sudden death. The role that "stress" (physical or emotional) may play varies with these different events. The relationship of stress to angina is well known and is beyond the scope of this report. The very term "angina of effort" indicates its intimate association with a multiplicity of stressful situations.

"Acute coronary insufficiency" is an arbitrary term used to designate those cases in which acute ischemia of the myocardium is more intense and prolonged than in the typical anginal attack, and yet is not severe enough to produce the classical signs and symptoms of myocardial infarction. Some prefer to use the term "coronary failure" rather than acute coronary insufficiency. Although myocardial necrosis is presumed to occur in these episodes, it is assumed that the areas of necrosis are localized and do not extend through the entire myocardial thickness as in the classical infarct. That is why some of these are classified as subendocardial infarcts. In approximately half of the cases this type of attack is entirely spontaneous, but in a few instances it follows such incidents as usual physical effort or excitement (Table II).[7] It is recognized that the grouping of cases into this category is not entirely satisfactory from either a physiologic or an anatomic point of view. Currently, however, it seems to serve as a convenient clinical designation for cases too severe to be classed with anginal attacks that fail to present the evidence needed to permit being designated as a myocardial infarction.

Table II—Activity at Onset of 100 Attacks of Coronary Insufficiency[7]

No unusual activity	56	Acute cholecystitis	1
Hemorrhage	10	Acute pancreatitis	1
Pulmonary embolism	8	Gastroenteritis	1
Unusual effort	6	Hypertensive crisis	1
Tachycardia	3	Postsympathectomy	1
Surgical procedure	3	Carbon monoxide	1
Excitement	2	Tobacco and alcohol	1
Meal	1	Addisonian crisis	1
Trauma	1	Asthma	1
Acute left heart failure	1		

The majority of cases belong in the category of acute coronary occlusion with myocardial infarction. At postmortem examination these may show only advanced coronary atherosclerosis, with marked narrowing of the arterial lumen, or the artery may be occluded by a thrombus superimposed on an atheromatous plaque. The ratio of the number of cases with atherosclerotic occlusion as compared with those with thrombotic occlusion will vary with the source of the data. In groups of cases from the clinical and hospital records, more than 70% will show thrombi. Among patients who died within minutes to an hour from the onset of the attack, the ratio is reversed with the instances with thrombi forming the minority. The latter group consists of those who died before reaching a hospital or clinic and are complied by medical examiner's and coroner's offices throughout the country: They constitute approximately 25% of all deaths from coronary occlusion. Despite the difference in the ratios of atherosclerotic to thrombotic occlusions in these two types of cases, there is similar statistical relationship, or rather a lack of one, to any preceding unusual physical effort.

In an extensive clinical study by Master of over 2,000 attacks of acute coronary occlusion, it was noted that in only 2% were the attacks preceded by unusual or severe physical exertion (Table III).[7] The evaluation of the precipitating events was based on the detailed histories of the activities of the patients during several weeks preceding the attack and up to its acutal onset. This is an important consideration because, once initiated, it may take hours or even days for the thrombus to develop to the point of occluding the artery. In the same study it was also noted that the time of occurrence of these attacks was statistically equally divided among all four seasons of the year and throughout the 24 hours of the day (Tables IV and V). One of the shortcomings of such a clinical investigation is that the type

Table III—Types of Activity of Onset of 1,347 Attacks of Coronary Occlusion[7]

Activity	Attacks	
	No.	%
Sleep	305	22.6
Rest	401	29.8
Ordinary mild activity	302	22.4
Moderate activity	117	8.7
Walking	198	14.6
Unusual or severe exertion	24	1.9
Total	1,347	100.0

of acute occlusion cannot be ascertained, and so uncertainty remains as to whether the attacks preceded by unusual effort were mostly of the thrombotic variety or not.

These findings are confirmed by similar analyses of consecutive autopsy cases of sudden death from coronary occlusion reported by various medical examiners' offices. This latter type of investigation has the advantage of permitting the accurate classification of the type of occlusion. In one such study carried out by the author (Spain and Bradess)[8] it was found that only about 2% of the cases of each type of coronary occlusion were preceded by an unusual physical exertion (Table VI). Only 13% of the atherosclerotic group and 17% of the thrombotic had physical activity of any sort preceding the attack; this difference is not of statistical significance (Table VII). There was also no difference in the ratio of male to female (9 to 1) between the thrombotic and atherosclerotic occlusions. Findings similar to those of Master were noted when the attacks of coronary occlusion were tabulated according to time of day and season of the year. Similar findings have been reported from an extensive study conducted in the New York City Medical Examiner's office. Thus it would appear that the incidence of unusual physical activity prior to an acute coronary occlusion, whether or not the attack had a fatal termination, is no more than would be expected by coincidence.

Despite this apparent lack of statistical correlation between unusual physical effort and the onset of acute coronary occlusion, there is an occasional case in which this relationship appears so intimate and dramatic that it cannot be entirely dismissed. This is also true of the occasional attack preceded by an overwhelming emotional upset. Anyone with extensive clinical experience can cite a case of an individaul who had been subjected to severe or acute psychologic or emotional upset and promptly developed acute coronary occlusion or in whom a "bridge" of signs and symptoms began with the stressful event and culminated in the acute occlusive episode.

Table IV—Seasonal Incidence in 1,151 Attacks of Coronary Conclusion[7]

Season	Attacks	
	No.	%
Winter	300	26.1
Spring	268	23.3
Summer	288	25.0
Autumn	295	25.6

Table V—Period of Day of Onset of 944 Attacks of Coronary Conclusion[7]

Period of Day	Attacks	
	No.	%
7 a.m. - 1 p.m.	267	
1 p.m. - 7 p.m.	220	
7 a.m. - 7 p.m.	487	51.6
7 p.m. - 1 a.m.	218	
1 a.m. - 7 a.m.	239	
7 p.m. - 7 a.m.	457	48.4

Table VI—Relationship of Type of Occlusion and Strenuous or Unusual Physical Exertion to Onset of Sudden Fatal Attack

Type of Occlusion	Cases	
	Total	No. with Strenuous Activity
Atherosclerotic occlusion	27	6 (2.2%)
Thrombotic occlusion	90	2 (2.2%)

Table VII—Relationship of Type of Occlusion, Sex, and Preceding Physical Activity

Type of Occlusion	Number of Cases	Male	Female	Number of Cases With All Degrees of Physical Activity Prior to Attack
Atherosclerotic	310	277	33 (10.6%)	50 (13%)
Thrombotic	100	90	10 (10%)	17 (17%)
Total	410	367	43	67

Foremost among the arguments used to demonstrate that psychologic or emotional stress is an important background factor in the development of coronary occlusion is the widely accepted impression that top business executives who are under constant high pressure because of greater job responsibility have a higher attack of acute coronary occlusion than other individuals of the same age. This concept, unfortunately, has been based on impressions and is entirely refuted by recent reports of carefully documented and controlled investigations. One such report, based on a five-year survey of over a thousand executives as compared with more than a thousand office workers in the same companies, concludes that the top management personnel actually had a lower incidence of attacks of acute coronary occlusion

than the office clerks (Lee and Schneider).[9] It is important to note that in this study both the occupational categories were in the sedentary group. Similiar analyses had come to erroneous conclusions because the sedentary executive groups were compared with physically active laboring groups. Another study made by the Medical Division of the DuPont Company concluded that no differences were found in the incidence of myocardial infarction among groups classified according to degree of job responsibility (Pell and D'Alonzo).[10]

Discussion

Practically all of the reports on the association of occupational physical activity with the incidence of coronary atherosclerotic heart disease suffer from biostatistical and measurement deficiencies. Therefore, the highly suggestive postmortem, clinical, and epidemiologic evidence relating sedentary occupations to a higher incidence of mortality and morbidity from coronary atherosclerotic heart disease requires further validation. Among these deficiencies is a lack of knowledge as to the exact level of energy expended in various job categories. Precise estimates of average on-the-job, daily energy expenditure cannot be applied to epidemiologic studies with the techniques available at present. Thus, physical activity in current studies can at best be described as light, moderate, or heavy. Evaluation of leisure time energy expenditure is most often scant or completely lacking. The pattern of activity relative to its rhythm and peak expenditures of energy are usually not known. Similar job classifications in different environments often have a different meaning as regards energy expenditure. Distribution of the various body forms as related to the type of activity engaged in is often not known and may prove to be an important determining factor in accounting for the differences in frequency of coronary atherosclerotic heart disease. A recent conference on methodology in the epidemiologic study of cardiovascular disease has set forth minimum criteria which may make these studies more meaningful and comparable with each other.[11] It is hoped that in the future studies will reflect the goals of this conference.

Evidence is accumulating that over the past 25 years, in this area of the earth, that there is a real and absolute increase in coronary atherosclerotic heart disease, especially in the white male. One of the most profound changes over this 25-year period in our way of life is related to the physical expenditure of energy. The marked increase in automobile transport, advances in automation and mechanization in industry, and preoccupation with television entertainment have markedly altered our behavior. These changes in our patterns of behavior certainly warrant further detailed investigation as to the role they might play in the current coronary atherosclerotic heart disease problem.

Summary

Current studies indicate that sedentary occupational categories are more closely associated with coronary atherosclerotic heart disease than the more physically active job categories. Sudden or excessive physical exertion prior to the onset of acute coronary occlusion does not appear with any frequency greater than one might expect from coincidence.

The frequency of coronary thrombosis in coronary atherosclerosis is unrelated to occupational physical activity or physical exertion.

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Stress, Intimal Hemorrhage, and Coronary Occlusion

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IT IS NOW firmly established that relatively severe grades of coronary atherosclerosis may exist in man without the production either of pathologic sequelae or of clinical signs or symptoms.[1] And it is equally certain that, more or less suddenly, such severe lesions may become clinically apparent with the development of angina pectoris, ventricular fibrillation with sudden death, and/or myocardial infarction. What are the mechanisms involved in the sudden conversion of these previously benign lesions to ones of casualty-producing proportions? And what, if any, are their implications in industrial medicine? In the present paper, I shall have little or nothing to say about such obvious mechanisms as lacerations with secondary thrombosis of the coronary arteries due to trauma or about cardiac ischemia from the physiological imbalances between coronary blood supply and myocardial demand that results from severe physical exertion. Instead, I will concentrate on the thesis that hemorrhage into atherosclerotic plaques is the trigger mechanism in the production of many of the clinical syndromes mentioned above.

The Origin of Intimal Hemorrhage

Prior to 1936, the subject of intimal hemorrhage received little attention from workers in the field of atherosclerosis; on the few occasions in which it was mentioned its origin was laid to reflux of blood from the arterial lumen into the intima through a defect produced by atheromatous ulceration.[2] In that year, however, I was able to demonstrate by serial sections that intimal hemorrhage is an intrinsic lesion within the plaque, that a definite layer of intact tissue lies between the extravasated blood and the lumen of the artery.[3] This intrinsic nature of intimal hemorrhage seems now to be generally accepted: When an intimal defect is seen in association with hemorrhage it should be interpreted as a "blow-in" into the lumen, not as a "blow-out" from the lumen.[4] The cause of intimal hemorrhage must therefore be looked for within the plaque proper; and here the presence of capillaries in close association with the hemorrhage offers an obvious explanation for the extravasated blood. This cause-and-effect relationship between capillary rupture and intimal hemorrhage seems also to be generally accepted. However, the reasons why capillaries in these locations rupture are still a matter of controversy. They will probably not be established until they can be studied under controlled experimental conditions, and this must await the production of the human type of atherosclerosis, with vascularized plaques, in susceptible animals. I am not too optimistic that this will ever be attained.

As Duff[5] stated several years ago: "It seems to be one of the special misfortunes of the human race that the development of atherosclerosis should be accompanied by the formation of a new capillary network in the thickened intima." A good deal of evidence is accumulating, however, that intimal capillaries rupture, in large part at least, because of high pressures within their lumens. These small vessels differ from capillaries elsewhere in the body in that many of them arise directly from main arteries in which the pressure of blood is unusually high, and this pressure must be reflected into the capillaries themselves.[3, 4] If this were not so, the capillaries would be collapsed and bloodless, which they are not. It has therefore been postulated that increases in arterial pressure will cause further strains upon the capillary walls so that eventually there will be imminent

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Presented in part at the Second International Meeting on Forensic Pathology and Medicine, New York, N.Y., Sept. 18-21, 1960.

By agreement, this article will also appear in the Proceedings of the Meeting under the title "Some Medico-Legal Aspects of Coronary Occlusion."

danger of their dilatation and rupture. As will be discussed later, elevations in arterial and intracapillary pressure may occur not only with persistent hypertension but also from bouts of transient hypertension from excessive physical exertion or emotional stress. Other factors, however, no doubt play a part, although a secondary one, in the rupture of intimal capillaries. If capillary fragility is increased, as it may be in a variety of conditions, rupture may be expected to occur at lower intracapillary pressures than when the lining endothelium is healthy. Nevertheless, some degree of positive intracapillary pressure (in excess of the transmural pressure) must be a prerequisite of rupture.

The Incidence of Intimal Hemorrhage

Now that pathologists are beginning to search for intimal hemorrhages, especially in the coronary arteries, it is surprising how many of them are coming to light. They can be demonstrated in several ways—some easy, others time-consuming. The simplest technique is to open the arteries with scissors longitudinally and then search the exposed surface with the naked eye, with a hand lens, or by transillumination after the outer coats of the artery are stripped away. Suspicious lesions thus revealed may then be confirmed by gross or histological section. In my opinion, opening of the coronary arteries longitudinally is far superior to the usual cross section technique for the demonstration of intimal hemorrhage; indeed, I suspect that many of these lesions have been missed in the past simply because they are often so small that they lie in the interval between crosscuts and are therefore not seen. In any event, I have recently used the longitudinal-opening method in autopsies on 100 consecutive fatalities in males of an average age of approximately 70 years and have found one or more intimal hemorrhages in the coronary arteries in 56%. Willis[6] found a somewhat lower percentage (36%) in the femoral arteries of men with an average age of 60 years.

An even higher incidence of hemorrhage of the coronary arteries can be demonstrated by clearing the vessels with chemical agents, as practiced by Winternitz and others[7] and by Durlacher and others.[8] Durlacher's results in this respect were particularly interesting: He found that recent hemorrhages were almost invariably present in persons dying suddenly of coronary artery disease, whereas they were consistently absent in cases of extracardiac sudden death. The medicolegal implications of this finding will be discussed later. A much simpler clearing technique is the cool-dry maneuver developed by Moffatt.[9] It is particularly valuable in the examination of large vessels like the aorta, revealing not only hemorrhages but the pattern of intimal vascularization as well.

The Association Between Intimal Hemorrhage and Coronary Thrombosis Using Different Pathology Techniques

Investigator	Pathology Technique	Cases of Thrombosis Studied	Intimal Hemorrhages	
			No.	%
Paterson	Serial section at close intervals	58	52	89
Horn and Finkelstein	Many sections	123	64	52
Nelson	Many section	10	9	90
Paterson	Many sections	34	21	62
Yater	Occasional sections	158(?)	26(?)	16
Hamilton	Clearing technique	14	12	85
Helpern	Complete serial section	17	14	82

Finally, intimal hemorrhages may be demonstrated readily by serial section technique, especially when good contrast stains (like Masson's connective tissue stain) are used. This technique is of value not only in demonstrating the intrinsic nature of intimal hemorrhages but also in showing the close association between the extravasations and superimposed coronary thrombi. This latter feature has been disputed over the past 25 years, but it now seems clear that the discrepancies in the results of individual workers can be laid to differences in technique. When thrombosed segments of arteries are sectioned serially, either completely or at very short intervals, the incidence of hemorrhage in association with thrombus is as high as 90%, whereas it falls to less than 20% if only occasional random sections are cut (Table).[4, 10]

The Effects of Intimal Hemorrhage

Up to this point, my paper has been concerned with observations that have been amply confirmed, that can therefore be regarded as facts. I shall now deal with various deductions, hypotheses, even speculations, that may be drawn from these observations. They fall into four categories:

1. Is high pressure in intimal capillaries the result of high arterial pressure?

2. Is high intracapillary pressure a major factor in the rupture of intimal capillaries and in the production of intimal hemorrhages?

3. Are intimal hemorrhages space-occupying lesions and therefore capable of stenosing arterial lumens?

4. Is intimal hemorrhage a cause of coronary thrombosis?

I shall deal with the first two questions together. Mention has already been made of the fact that many capillaries within atherosclerotic plaques arise from the lumens of main arteries. Since these capillaries contain red cells and are sometimes dilated, the pressure within them must be as great as the pressure in the arterial lumens.[4] If this were not so, the transmural pressure (which is a reflection of the arterial pressure) would collapse these small vessels. It follows from this that leakage of blood from capillaries will occur only *when the intracapillary pressure is greater than the arterial pressure*—at any one point in a plaque and at any one phase of the cardiac cycle. This deduction is not as fantastic as it may seem: I have described elsewhere in detail how, by the application of Bernoulli's principle, the lateral pressure on a stenosing atherosclerotic plaque may be greatly reduced during a period of high coronary blood flow from strenuous physical exercise and how, if intimal capillaries lie in this area of reduced pressure but arise proximally at wider points in the artery where the lateral pressure is high, a wide discrepancy between capillary and arterial pressure might develop.[4] In these circumstances, capillary dilatation and rupture may well occur; and it should do so as the direct result of physical exertion! A recent report from our laboratories offers some factual evidence in support of this deduction: Intimal hemorrhages of the aorta, as demonstrated by the cool-dry technique, were significantly more numerous in hypertensive persons than in normotensives ($P < 0.001$).[11] If persistent hypertension can produce this effect, it is reasonable to assume that transient bursts of elevated pressure from physical or emotional stress will do the same, although perhaps to a lesser degree.

Naturally, these views have not gone unchallenged. Master,[12] for example, has stated that there is no evidence that the high arterial pressure of hypertensives is reflected into intimal capillaries; he prefers to regard intimal hemorrhage as a fortuitous event in the natural course of atherosclerosis. Texon[13] follows a similar line, stating, "It appears physically impossible for a transient rise in coronary blood pressure resulting from exertion to be transmitted in any significant degree through the microscopic lumen (5 to 15 microns) of the intimal capillaries arising from the involved coronary artery." I refer each of these writers to my remarks in the paragraph above as well as to a more detailed account of the biophysical principles involved in capillary rupture which have been published elsewhere.[4]

So much for my first two questions. An answer to the third, the query regarding the space-occupying properties of intimal hemorrhage, can be given by applying similar arguments. The addition of blood to a plaque must increase its bulk, the order of magnitude of the increase depending directly upon the quantity of blood extravasated. Intimal hemorrhages are thus space-occupying lesions; but, what is more important, the displacement should occur inwardly because of the restraining influence of the great tensile strength of the adventitia.[7] Inward displacement, therefore, must mean that the pressure within the hemorrhage is greater than the pressure in the arterial lumen. Stenosis of the arterial lumen, to a degree, must therefore inevitably occur from intimal hemorrhage. If the hemorrhage is of only moderate size, the anginal syndrome would be expected to result; but if large, coronary occlusion with sudden death or myocardial infarction should occur. Wartman[14] and more recently Helpern and his associates,[10] have stressed this latter effect; indeed, there is reason to believe that it may be almost as important as thrombosis in occluding the lumens of sclerotic coronary arteries.

My final question concerns the significance of the high level of association that has been found between intimal hemorrhage and thrombi in coronary arteries and shown in the Table. Is this association fortuitous, or is the hemorrhage the cause or the result of thrombus deposition? For many years, I have held the opinion that hemorrhage and thrombosis represent a cause-and-effect relationship, and I see nothing in the recent literature to make me change my view. The factor of coincidence is improbable, since an association of 80 to 100% has been reported by at least five different observers, working independently (Table). Secondly, because intimal hemorrhage can be demonstrated so often in coronary arteries *without* thrombosis, it cannot be regarded as a secondary intimal change resulting from the presence of an occluding mass in the external lumen—as a form of hemorrhagic infarction of the intima as suggested by Hamilton.[16] By a process of elimination, then, one must deduce that a hemorrhage into the intima has thrombogenic properties, that it probably acts in a way similar to that of certain inflammatory lesions in the walls of arteries which induce thrombosis

(e.g., thromboangiitis obliterans and polyarteritis nodosa). Certainly, intimal hemorrhages are disruptive lesions that tear tissue; and in doing so they may well release thromboplastic substances which, if conditions of blood flow are suitable, initiate the primary deposition of platelets and fibrin. But there is one additional feature of coronary thrombosis that must be understood clearly: It is now very evident to pathologists that many coronary thrombi grow to occlusive proportions quite slowly after they are first initiated.[4, 17] Indeed, as shown by the degree of organization of various parts of the thrombus, many hours elapsed between the primary deposits and the laying down of the final clumps of fibrin and platelets which presumably give rise to the signs and symptoms of occlusion. In these circumstances, the activities of patients at the moment of clinical occlusion (as described recently by Master[21]) have no connection with the causes of primary thrombus deposition.

The Medicolegal Significance of Intimal Hemorrhage

The crux of the matter lies in the validity of the proposition that excessive physical exertion (or emotional stress) may so elevate blood pressure and so increase coronary blood flow that intimal capillaries in preexistent semistenosing atherosclerotic plaques will rupture and produce intimal hemorrhages. If this proposition is accepted, certain deductions of medicolegal importance may be made.

Depending on the size of the intimal hemorrhage (and thus on its space-occupying properties) and on whether or not secondary thrombosis occurs, the obstruction to coronary blood flow will be moderate or severe, immediate or delayed. Hemorrhages of only moderate size, if uncomplicated by thrombosis, should produce nothing more than angina pectoris—unless they are multiple, when perhaps a cumulative effect on coronary flow may result (as in Durlacher's[8] cases). On the other hand, large hemorrhages should result either in myocardial infarction or in acute coronary insufficiency with sudden death. In either case, the time relationships between the causative stress and the onset of clinical symptoms should be more or less immediate.

A clinically silent period of many hours or days may elapse, however, in those persons in whom a stressful incident causes intimal hemorrhage with resultant coronary thrombosis. The reason for this, as has been explained previously, is that most coronary thrombi are built up slowly in layers. Thus it is wrong to state that unusual stress could not have been responsible for the original intimal hemorrhage and thrombus deposition in a person who develops his first occlusive symptoms while at rest, long after the stressful incident. If the age of the oldest hemorrhage and thrombus agrees with the time interval between stress and death, one can state that there may have been a relationship, but this should be qualified by the admission that other forms of stress, occurring about the same time although not related to work, may have been equally at fault. Dock[18] has listed these nonoccupational stresses and asserts: "If we are to assign values to all the stresses, physical and mental, of all the employees who develop heart trouble, it is unlikely that occupational causes contribute 10 per cent of the total.

A pathologist may be called upon, nevertheless, to give expert testimony on the relationship, if any, between a particular unusual work stress and a fatal coronary occlusive incident, either immediate or delayed. Assuming that a very searching pathology examination of the coronary arteries has been made and all extracardiac causes of death eliminated, I suggest that the following views might be expressed. They are the same ones that I made on a previous occasion, with slight modifications.[19]

A. *For persons in whom unusual work stress is followed almost immediately by clinical occlusion and death*
   1. When no acute lesion such as hemorrhage or thrombosis is found and only an extreme grade of atherosclerotic stenosis is demonstrated, death should be attributed to a severe physiological imbalance between coronary supply and myocardial demand, the direct result of the unusual stress.
   2. When a massive intimal hematoma occludes the lumen of a main coronary artery and it is histologically recent in its entirety, death should again be attributed to the unusual effort which resulted in the rupture of intimal capillaries with severe and rapid bleeding into a preexistent atherosclerotic plaque.
   3. When a coronary thrombus is found and some part of it shows histological evidence of organization (and must therefore be at least 24 hours old), the relationship between the unusual effort and death must be regarded as coincidental.

B. *For persons in whom clinical occlusion and death is delayed for many hours after an unusual work stress*
   1. When an occluding thrombus is found and if the age of its oldest part (and of the initiating hemorrhage) agrees with the

interval between the unusual effort and death, one can state that there may have been a relationship between the two; but this should be qualified by the admission that any other stressful episode, not related to work but occurring about the same time, may also have been responsible.

These are my specific recommendations; but as Sir Winston Churchill said in 1948, "It is always more easy to discover and proclaim general principles than to apply them."[20] And this is especially so when we admit that it is often difficult to ascribe a definite age to a particular intimal hemorrhage. It seems to me therefore that we should look elsewhere for a solution to this difficult matter, a solution that will be fair to both employee and employer. Specifically, I suggest that consideration be given to a recommendation that was made at the First Wisconsin Conference on Work and the Heart. In a modified form, it was as follows: "That it would be best to develop some type of broadened insurance scheme to cover deaths from coronary occlusion in all individuals engaged in stressful occupations, admitting that in some instances we just do not know whether or not work is responsible. We would thereby avoid subsidizing the families of some workers dying of coronary disease simply because death occurred at work, whereas others are deprived of such benefits because death did not occur at work, although there may have been stress on the job that played a prominent role in causation."

But until such a scheme is brought into force (and it may be an expensive one), I suggest that the rules listed earlier in this section be followed as closely as possible.

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Problems of Cardiac Disability and Death Claims

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THERE is widespread conviction that the statutes and court decisions relating to heart cases are in great confusion and uncertainty, that heart claims are growing in number and importance, and that it is time for a complete reexamination of the law in this area. Vast amounts of money and talent have been expended to determine the medical relationship between effort and the onset of symptoms of heart disease, but little or nothing has been done to determine the optimum legal procedures and methods which should be used to adjudicate the legal claims of those who sustain heart attacks while on the job and elsewhere. This is a preliminary report of findings of a research project* whose purposes include a correction of this one-sided approach.

Procedures in Making the Study

The basic step in any investigation is, of course, finding the facts. One method of gathering information for my research project has been extensive personal interviews and conferences with knowledgeable persons and groups in the cardiac field. Many leaders in industrial medicine, workmen's compensation, and insurance law have been contacted, and the authors of past studies on various aspects of the cardiac problem have been approached for information.

A second major phase of the research project has consisted of collecting published material on the subject, and obtaining written reports and comments from those who deal with the legal, medical, industrial. and other aspects of the cardiac problem. Effort has been made to contact as many as possible of the leading lawyers and other experts who might be expected to have special knowledge in this field.

Communications soliciting information and expressions of opinion have been sent to all the workmen's compensation commissions in the United States and to selected commissions in Canada, as well as to 350 of the larger corporations in the United States in such diverse fields as merchandising, transportation, public utility service, life insurance, and banking. In addition, the medicolegal centers at all the American universities which have such centers have been approached, as have 200 insurance companies which operate in the workmen's compensation field, a number of labor unions, and several federal agencies. A remarkably large percentage of these inquiries have elicited replies, and many of those answering did independent research or at least reviewed their own files before formulating responses. The study has thus been assisted by thousands of man-hours of expert labor on a purely voluntary basis.

A third step in the investigation has been the preparation of an analysis and evaluation of current law and practice with respect to the processing of cardiac claims. This analysis, which is still going on, is being made on a state by state basis. I and members of my research team collate and analyze the statutes, the decisional law, and, so far as possible, the practices of each state. We then mark uncertain areas for more intensive investigation, and prepare comments and criticisms. This phase of the study is the most important and also the most time-consuming, since a great volume of statutory law and litigation in 50 states has to be examined.

After a draft of the analysis of the law in a particular state has been completed by the research team, we submit it for critical scrutiny

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From an address before the Section of Insurance, Negligence and Compensation Law of the American Bar Association, Washington, D.C., Aug. 30, 1960. This somewhat abbreviated version is reprinted with permission from *Insurance Law Journal*, in which the full text appeared in October, 1960.

Received for publication November 7, 1960.

*The project entitled "The Legal Basis for Awards in Cardiac Cases" is supported by a grant from the National Heart Institute, United States Public Health Service.

to persons having special familiarity with the situation in the state in question. The general procedure is to send the draft to the workmen's compensation commission of the state, to the governor's office, to a prominent attorney identified with heart cases as a claimant's representative, and to a prominent attorney identified with heart cases from the side of the insurance carrier. As would be expected, persons intimately familiar with the practice in a particular state can provide insights which cannot be derived from a mere examination of the published decisions, and can correct misconceptions which might flow from analyzing the decisions without a knowledge of the practical operations of the law in the state.

Incidentally, at this stage, only about half of our drafts of state law have been reviewed by the experts in the various states. Thus, as to many states, the report has not yet been fully processed.

The final step in the study, which we will begin after revising the various state drafts in the light of the comments by the experts in each state, will be a review of the state drafts with the purpose of determining the state of the law in the country at large, and the preparation of a report setting forth the over-all national picture, with any conclusions or recommendations deemed to be appropriate.

Principal Factors in the Decision of a Cardiac Compensation Case

If there is one point which now emerges with great clarity from the continuing study, it is the difference in the decisions in cardiac cases even where the facts are similar or virtually identical. It is this variation in decision, incidentally, which perplexes many of our friends in the medical profession. My staff and I have come to the conclusion that there are a group of principal variables which, dependent upon how they combine in a particular case, will be largely determinative of its outcome. It should not be assumed, of course, that all of the variables to which I will hereafter refer are necessarily restricted to heart cases. Some of them are applicable to all types of compensation cases, and even to other areas of the law. As will hereafter appear, however, they do have peculiar application to the cardiac area in view of the large number of death cases litigated.

To illustrate what I have in mind, let me put to you the hypothetical case of an employee, Mr. X, who, in the course of his morning's work, lifted a 100 lb sack of flour at the mill where he was employed. Weakness and shortness of breath followed the lifting, as did some chest discomfort. Late that evening he died of a cardiac involvement. Is Mrs. X, the widow, entitled to compensation benefits?

The Language of the Compensation Statute—At first glance, it might be assumed that the rights of Mrs. X would turn simply on the provisions of the compensation statute in the state where the incident happened. That is, is the statute framed to cover all "personal injuries" arising out of employment or is an "accidental injury" required?

Upon closer analysis, however, it becomes clear that the outcome of a cardiac claim cannot be gauged accurately by the use of such a simple common denominator as the wording of the compensation statute. Although the language of the statute will be of some importance, the construction given to the relevant sections by the courts of the state will be of much greater significance.

The Interpretations of the Courts: The Usual and Unusual Strain Rules—The approach of the courts, in applying a compensation statute to heart cases, varies a great deal from state to state, although it is possible to put states with similar approaches into general categories. These varying approaches have produced different judicial attitudes with respect to many questions, as, for example, the question whether the strain of a workman's normal duties will support a compensation award when he suffers a cardiac disability following the performance of such duties.

While it is somewhat of an oversimplification, it may be said that, in general, there are two basic judicial approaches to this question: the "usual strain" rule, and its opposite, the "unusual strain" rule. The "usual strain" rule will support a compensation award even though the cardiac incident followed performance of the employee's regular duties, while the "unusual strain" rule will require something over and above those normal duties.

An interesting point to be noted is that the "usual strain" rule and the "unusual strain" rule are both doctrines which the courts themselves have worked out, and have no special relation to the wording of the particular compensation statute. The compensation acts of Tennessee and Missouri, for instance, are both phrased in terms of an injury by accident being compensable. However, construing its statute, Missouri has ruled that "unusual" strain is necessary, whereas Tennessee, interpreting the similar wording of its law, has held that "usual" strain will suffice, provided, of course, that a causal connection between the strain and the cardiac incident is established.

There are, moreover, variables within variables. The courts of the various states differ not only in their views as to whether "usual" or "unusual" strain is the criterion to be applied but also in the meanings they give to "usual" and "unusual" strain. The result is that, even if State A and State B have identical compensation statutes, and even if the courts of both have embraced the "unusual strain" rule, the decisions actually arrived at in each state may be quite different. State A may feel that any strain over and above that required in sedentary tasks qualifies as "unusual," whereas State B may regard nothing short of a Herculean effort or calamity as "unusual." Thus, in a New York case,[1] the exertion of using the subway stairs while carrying a brief case was held to be unusual strain, while in North Carolina much heavier exertion was held to be usual and insufficient. In the North Carolina case I have in mind,[2] a fire chief pulled 700 feet of heavy hose out of a fire truck, ran through a building up to the attic, and assisted other firemen in pulling a hose through a window. A heart attack occurring immediately thereafter was held to be noncompensable, the strain being regarded as usual.

Still other subcategories can be found within the unusual strain rule. In some states, for instance, the rule is applied on an occupational or trade basis rather than on an individual basis. That is, some jurisdictions look to what is unusual for mill workers or truck drivers as a class, whereas others focus on the question whether the strain was unusual for the particular mill worker or the particular truck driver in question. Pennsylvania would be illustrative of the jurisdictions leaning toward the occupational approach, while Maryland is typical of the states emphasizing the individual claimant's status. Again, some jurisdictions, like New York and Puerto Rico, have an unusual strain rule but hold it to be satisfied where the act in question is at all strenuous, even though it is normal for the employment and the particular employee.

It is apparent, therefore, that the chances that our hypothetical widow, Mrs. X, can recover for her husband's death will be much affected by the decisional gloss which the courts have placed upon the general language of the statute applicable to her case.

Impartial Medical Panels and Standardized Medical Criteria—Once the widow's claim reaches the hearing stage, several other variables will come into play, depending again on the particular jurisdiction involved.

In some states, the medical aspects of the widow's claim will be submitted to an impartial physician appointed by the workmen's compensation commission or even to a panel of impartial physicians. In many other states the employment of such neutral physicians is not authorized, or, if the authority to employ them exists, they are not used as a matter of practice. Particularly if there is a sharp conflict in the testimony of the medical witnesses produced by Mrs. X and those produced by the insurance carrier—and there usually is such a conflict—the decision in the case may well turn on the report or the testimony of the impartial physician or panel in a state where such an impartial system exists. On the other hand, in jurisdictions not utilizing such a system, the compensation commission will have to decide the case, as best it can, by choosing between the conflicting testimony of the medical experts presented by the parties.

The claim of Mrs. X may also be affected by the use of standardized medical criteria applicable to cardiac cases, such as fixed time limitations for the appearance of symptoms. While the use of standardized medical criteria is relatively new and experimental, Mrs. X would be confronted with such criteria in Utah and Washington. Both of these states, with the aid of medical advice from their local heart associations, have established minimum medical criteria for use in the processing of cardiac claims, and a number of other states are examining into the possibility of doing so.

If a claim does not measure up to the criteria, the claimant is not automatically ruled out, but his chances of success are minimal. If the claim does fit within the criteria, the prospects of a successful outcome for the claimant are materially enhanced. If the claim of Mrs. X arises in a jurisdiction which has not adopted minimum criteria, the benefit or detriment of such standards would, of course, not be a consideration in her case.

Autopsies—Another medical consideration which may have a material impact on the claim filed by Mrs. X is the over-all policy of the jurisdiction with respect to the holding of autopsies in compensation cases.

In State A, autopsies may seldom, if ever, be used, whereas State B may have a uniform practice of paying great heed to autopsies in cardiac cases. As a consequence, State A may treat the claim of Mrs. X as simply falling within the generic category of "heart attack" cases, whereas State B, fortified with the detailed information flowing from the autopsy, may deal with the case on the basis that it is one specific kind of heart case such as a fresh

or an old coronary occlusion with an old or a fresh myocardial infarction. In states where autopsies are seldom or never used, the medical evidence and the opinions of the courts tend toward vague general discussions, whereas, in states where autopsies are regularly employed, the medical evidence and the opinions direct themselves specifically to a particular kind of heart disease and its causative factors.

At least at this point in my study, I am unable to say whether the attitude of a state toward the use of autopsies bears any relation to the number of cardiac cases in which claimants are successful. While it might be supposed that a claimant would find it more difficult to succeed in a case where an autopsy report is presented against him, this is not necessarily so. Thus, in a recent New York case[3] the court upheld an award made by the compensation board despite a coroner's testimony that an autopsy was not possibly susceptible of the conclusion that a particular accident precipitated a coronary attack. On the other hand, some states seem to treat autopsy findings as virtually incontrovertible. It seems fair to say, however, that in states where autopsies are widely used, the battle of causal relation is fought on a more intelligent plane.

The use of autopsies in cardiac cases in a given state seems to depend upon a complex of factors. These include the presence or absence of a statute specifically authorizing the use of autopsies in compensation cases and the pressure or lack of pressure by attorneys for their use. The conservatism of the bar and its tendency to proceed only in paths which have been well worn by prior use is evident. In states where autopsies have not been used in the past, there seems to be a reluctance to argue for their use today.

Death Certificates and Coroner's Findings—The view which a state takes as to the significance of death certificates may materially affect the outcome of a cardiac case. The same is true as to the attitude of the state on testimony given by a coroner or medical examiner.

In jurisdictions such as Missouri, New Jersey, and Ohio, a statute declares that death certificates are prima facie evidence of the facts contained therein. This means that the cause of death, as stated in the certificate, is established prima facie merely by the introduction into evidence of the death certificate. A number of other states treat the certificate as evidence only of the fact of death and not its cause. As would be expected, in some of the states which treat the certificate as prima facie evidence of the cause of death, the findings recited in the certificate appear to play a significant role in the ultimate decision. On the other hand, in many states, the opinions make no mention at all of death certificates, and in still others scant weight is given to them and they are easily discredited by medical opinion adduced at the hearing.

A similar lack of uniformity in approach has been observed on the question of the admissibility of, and the weight to be attached to, the reports or testimony of the medical examiner or coroner. Many states flatly exclude such reports as inadmissible hearsay, others admit the reports as evidence, and in still others such an official often appears as a medical witness.

Preference for the Specialist or the Treating Doctor—Certain jurisdictions seem to have a predisposition to give greater weight to the testimony of a certain class of physicians, such as the cardiac specialist or the physician who actually treated the patient. By a predisposition, I mean that the courts in such jurisdictions seem to start off with an assumption, sometimes explicit but often tacit, that the expert of the particular preferred class is the witness to whom deference should be given.

Presumptions—The presence or absence of presumptions is a factor which may have an important effect upon the determination of the claim filed by Mrs. X. The courts of many states will presume, prima facie, that the death of a workman arose out of his employment when he is found dead at a place where the duties of the employment reasonably required him to be. Thus, if a workman dies of a cardiac ailment, within the time and space limits of his employment, and there is no evidence as to the actual facts surrounding his death, his widow may nonetheless be able to recover upon the strength of the presumption. Mississippi, Missouri, and many other states have such a presumption by case law, and Massachusetts has it by virtue of an express provision of its compensation act.

Although this presumption is rebuttable, it may be of great help to a claimant in a cardiac case. Indeed, some states seem to treat the presumption as evidence to be weighed on the claimant's side against the proof adduced by the insurance carrier. By the same token, in a state which does not have the presumption, its absence may play a major part in the denial of a claim in a case where a workman dies on the job of an unwitnessed heart attack. In such unwitnessed cases, it is often impossible, absent a presumption, to show the necessary nexus between the employment and the cardiac incident.

The presence of presumptions does not, of course, always benefit a claimant. Thus, some states appear, by case law, to have developed what almost amounts to a presumption that a heart attack is due to the natural progress of disease and does not arise out of employment. Expressions of this nature can be found in the opinions in New Jersey and North Carolina. Other jurisdictions seem to reach somewhat the same result by a kind of judicial notice. That is, the courts in such jurisdictions reinforce decisions wherein compensation is denied by citing, as if to take judicial notice, the fact that heart disease is the nation's number one health problem. The implication of such statements is, presumably, that the death or disability occurred by virtue of an ordinary disease process rather than because of employment.

On the other hand, in some states the claimant has benefited from a sort of res ipsa loquitur doctrine in cases showing a fact pattern of prior good health or prior good work record, a work-connected incident, and an immediate decline in health culminating in a cardiac death or disability. Such a pattern has been held to "bespeak causality," that is, the progression of events was such that a causal relation between the work and the cardiac incident could be inferred, sometimes even without medical testimony on the point. Such decisions have been rendered in Pennsylvania and elsewhere.

It can readily be seen, therefore, that the outcome of a cardiac claim may depend, in large measure, upon what presumptions, or judicial attitudes akin to presumptions, prevail in the forum where the claim is litigated. These presumptions or attitudes run from one end of the scale to the other, from the presumption that a cardiac disability was caused by the employment to the presumption that it was the natural result of an underlying pathology.

Attitude of the Reviewing Court—Judicially created presumptions are one reflection of the attitude of the reviewing courts in cardiac cases. The presence or absence of such presumptions, and the effect given to them, are likely to constitute a good index of the philosophy in cardiac cases of the reviewing court of a particular jurisdiction.

There are, however, other factors in the process of court review which have significance as well. One of these factors is the general approach of the court toward judicial review of administrative determinations in compensation cases.

The theoretical formula regulating the scope of judicial review does not vary very much from state to state. While the verbalization of the formula may differ somewhat, it comes down pretty much to the same test in all jurisdictions, namely, is the determination supported by substantial evidence? But in the application of this test there is considerable variation from state to state.

Most courts typically dispose of an appeal in a cardiac case by holding, in a brief opinion, that the determination of the compensation commission was supported by substantial evidence. On the other hand, the opinions in some states habitually contain large extracts from the medical testimony presented before the commission, almost as if the court felt compelled to prove to the skeptical that the record did in fact contain the requisite substantial evidence to support the determination. A few of the courts which use this approach seem to go beyond the traditional scope of review and actually weigh the medical testimony. It is not unusual in such states to see the administrative determination reversed and a claim dismissed, particularly where the medical testimony was couched in terms of a "possibility" rather than a "probability" of causal connection.

In addition to these differences in judicial attitudes in passing on the facts, there is quite a divergence in view in approaching the law in cardiac cases. In some jurisdictions, the legal questions are treated simply and summarily and seem virtually to merge into the over-all factual question whether the work caused the cardiac involvement. In many states, however, the opinions are replete with discussions of legalistic issues of a highly technical nature, such as whether the commission correctly construed the basic liability section of the compensation statute, whether it correctly applied the "unusual strain" rule or the "usual strain" rule, as the case may be, whether an accidental cause is required or only an accidental result, and so on. In jurisdictions which have exalted the significance of such inquiries, the courts are concerned with questions of statutory interpretation in almost every decision.

Those of you who are intimately familiar with the problems in heart cases can doubtless add other factors to my list of the variables which act and interact to produce the decision in a workmen's compensation case. We can probably all agree, however, that it is of great importance to segregate these variables so that intelligent analysis can be made of each. There has been far too much generalizing about the heart cases and too little examination into the specific factors which combine to produce the decisions in such cases.

Of course, listing the variables and seeing how they operate in each state is only the beginning. The job which lies ahead of the study is to try to come up with some useful suggestions and recommendations for dealing with these variables in the interest of improving the decision-making process in a manner which will be just to all parties concerned.

The Interplay of Compensation Decisions with Other Areas of the Law

The emphasis which I have given to the intricacies involved in the processing of cardiac claims in the compensation area should not be taken as an indication that the law of workmen's compensation has a monopoly on the legal questions surrounding cardiac incidents. On the contrary, while workmen's compensation is undoubtedly the most significant area where the cardiac battle is being waged, it is by no means the only one.

As a matter of practical limitation, in order to avoid dissipating our energies over too broad a field, my staff and I have concentrated largely upon workmen's compensation. Nevertheless, we are also investigating, on a less intensive basis, the ramifications of the cardiac question in such areas as common-law negligence, Federal Employers' Liability Act, Longshoremen and Harbor Workers' Act, accident insurance, life insurance, and public and private pensions. In these fields we have found problems of statutory interpretation, common-law negligence, and contract law which are unrelated to the law of workmen's compensation. However, in view of the fact that cardiac cases in the compensation field have been litigated in much larger numbers than anywhere else, it is not surprising that we have also found compensation decisions influencing other areas of the law. That is, there is a discernible tendency for lawyers and judges to apply the cardiac decisions in the compensation field to cardiac cases arising in entirely different legal environments. The question of the desirability of such interplay is one of the issues which the courts are just now beginning to face.

Illustrative of what I have in mind is the emerging interchange between cardiac cases in the compensation field with pension law cases. In several states, the question has already arisen as to whether the law as evolved in compensation cases should have any bearing on the ultimate decision in a pension case growing out of a cardiac incident.

For example, in a case decided in California,[4] the widow of a fireman sought a mandamus directing the trustees of a pension system to award her the special benefits which were payable when an employee died as the result of his work. The widow had previously recovered benefits under the workmen's compensation law on the basis that her husband's fatal heart attack was work-connected. The pension board argued that the prior award was not res judicata in the pension litigation. The board pointed out that, under the compensation statute, it was presumed, in the absence of contrary evidence, that heart trouble which manifested itself during a fireman's employment was caused by such employment, but that the presumption had no application to a pension claim. Despite this difference in the burden of proof, the California Supreme Court ruled in favor of the widow on the theory of res judicata.

A less striking but still important interplay between compensation law and pension law is evident in Massachusetts. The prevailing view in Massachusetts was that the word "accident," as used in a public retirement plan, had a narrower meaning than the term "personal injury" as used in the workmen's compensation act. However, in a case decided about two years ago,[5] the Supreme Judicial Court ruled that an amendment to the pension statute substituting the phrase "personal injury" in place of the word "accident" had extended the coverage of the pension law. Relying in part upon the broad meaning given to the term "personal injury" by the compensation decisions in Massachusetts, the court held that a heart attack suffered by a city engineer the day after an argument at work was a "personal injury" within the intendment of the pension legislation. This was true even though the engineer had a history of heart disease and had been advised to avoid excitement.

The courts are not all in agreement, however, in equating compensation law and pension law terminology. Illustrative of a different approach is a recent New York decision[6] wherein a claim was filed against the Comptroller of New York State in his capacity as head of the state employees retirement system. A 59-year-old forest ranger had suffered a mild cardiac insufficiency, followed by a myocardial infarction which hospitalized him for several weeks. Thereafter he returned to his usual duties, and, approximately a year later, he collapsed and died after fighting a fire on a steep hill. His widow applied for benefits under a statute providing for an "accidental death benefit" upon the death of a member of the retirement system "as the natural and proximate result of an accident sustained in the performance of duty."

The Comptroller found that the forest ranger

died of a coronary occlusion secondary to a preexisting condition of coronary arteriosclerosis and myocardial infarction, and that his death was "precipitated" by his fire-fighting activities. Nevertheless, the Comptroller ruled that the case did *not* constitute a compensable accident within the meaning of the Retirement Law. The New York Court of Appeals affirmed his determination. In that court's view, the Comptroller was not compelled to follow contrary decisions which had been reached on similar facts under the workmen's compensation law. Stressing the different social philosophy underlying retirement and compensation laws, the court commented:

> . . . There is quite a distinction between the situation of an ordinary workman and a civil servant; and the Comptroller is dealing with a fund quite different than insurance designed to protect workmen and shift the burden imposed by industrial accidents to industry as a whole. Inherent in this is the suggestion at least that the Comptroller . . . is not bound to accept the tests usually applied in compensation cases.

These decisions in California, Massachusetts, and New York are significant not so much for the conclusions reached by the courts therein but rather as illustrations of a problem which appears to be of growing importance. If the courts are going to transfer holdings in compensation cases over to the pension field or to some other area of the law, it seems reasonably clear that they should do so as a matter of deliberate, reasoned choice, rather than merely as a matter of mechanical application of a compensation law definition. Interplay of this sort between one field of law and another is neither good nor bad in itself. It all depends on whether the transplating of the principle is done in a rational manner, with full regard to any divergences which may exist in the social policy considerations underlying the different areas of the law.

Conclusion

What I have said here today has not been designed to present any recommendations as to improvements in the law in the cardiac field. As I mentioned at the outset, this study is still far from completed, and it would be presumptuous to state conclusions before the evidence is all in. What I have tried to do is to identify some of the forces which are operating to produce the decisions in the heart cases in the compensation field and to suggest some of the questions presented by an emerging tendency to project the compensation decisions into other areas of the law.

I strongly believe that clear identification of the forces which bring about the decisions in the cardiac cases is the necessary preliminary to evaluating those decisions and recommending improvements in this field of the law.

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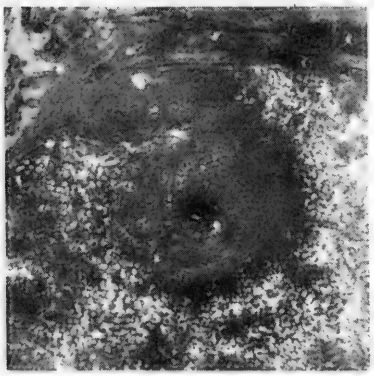

Correction

The illustration shown as Figure 8 in the article "Tuberculosilicosis: A Study of Its Synergistic Mechanisms" by Paul Gross, M.D., Marian L. Westrick, Ph.D., and James M. McNerney, M.P.H., on page 573 of the December, 1960, issue of this journal was incorrect. The correct Figure 8 is shown here at the left. Reprints with the correct illustration may be obtained from the authors, Industrial Hygiene Foundation, 4400 Fifth Ave., Pittsburgh 13, Pa.

Heart Disease and Compensability Under Workmen's Compensation

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CURRENT cardiology stresses the fact that the cardiac, with relatively rare exceptions in the most severe cases, not only can but should work. Experience recognizes the transient character of some heart disease. Employment in useful and remunerative activity helps nearly every cardiac patient. Industry would profit from this storehouse of skills, and society's burdens of relief and welfare would be lightened.

Notwithstanding assurance of their physicians, heart patients have difficulty in obtaining employment. Part of this is due to their own fear of further attacks, but more frequently because of the reluctance of employers knowingly to hire them. This is due to fear of increased workmen's compensation insurance costs.

Test of Compensability in California

Any disease, disability or death which arises out of and occurs in the course of the employment in California is compensable.[1]

While the extent of liability varies, it is immaterial so far as basic compensability is concerned whether industrial stresses created primary heart damage or aggravated a preexisting disease.[2] The test of liability is the effect of a specific injury upon the employee, not what it would have been on a healthy person.[3] If the prior pathology is active and progressive, is producing symptoms, and superimposed trauma aggravates the underlying lesion to produce disability, industry is liable only for the effects of the aggravation.[4] Should death result, there is no apportionment. Industry is wholly liable.[5] Where the preexisting pathology is asymptomatic prior to injury, and industrial injury produces disability which did not exist independently of injury, the entire condition is compensable.[6]

Presented at the Annual Meeting, California Heart Association, Long Beach, Calif., May 23, 1959.

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Present Test of Compensability

It is self-evident that disability due to the normal progression of a disease occurring in an employed person is not an injury and is not compensable.[7]

The Labor Code does not require that the industrial cause be an "accident" in the conventional sense of that which is sudden, usually violent, and often external. However, the early heart cases required that the heart difficulty be precipitated by unusual stress or overstrain to distinguish that case from the normal progress of the disease.[8] Because the test of compensability has been a question of fact in each individual case, a wide variety of causes have been found to give rise to compensability, some of them seemingly original in that case.[9]

Importance of Medical Testimony

Questions of pathology involve matters known only to medical experts. Their opinion alone is competent to establish cause of a heart episode.[10] The Law makes no distinction between "medical experts." The general practitioner before the court is the legal equal of the specialist. The weight to be given to conflicting medical opinion is a question for the triers of fact.[11] These are laymen, possessing no more than superficial familiarity with medical problems, hence can be misled.

The Labor Code provides that reports of attending or examining physicians are receivable in evidence in lieu of their oral testimony.[12] The referee rarely sees the medical authors. Their reports must speak for themselves. If a report is incomplete in its factual contents, its professional value may be over- or understressed.

Contents of an Adequate Report by Specialist

In studying the relationship of physical or emotional strain to the heart, certain fundamental facts must be considered: (1) What was the condition of the heart prior to the claimed

injury? (2) How severe was the injury, and what part of the body was involved? (3) What was the type of injury—crushing, penetrating, strain? (4) What symptoms were present at the time of the injury and shortly thereafter, and what was the time of their appearance? (5) How many of the examination findings actually preexisted? (6) Was an underlying, preexisting lesion present, and, if so, was it aggravated? (7) If strain is accused, precisely what was being done at the time? (8) Was the activity normal or unusual for the patient? (9) What was the effect upon the patient? (10) What extracardiac complications exist in the patient and what are their effects?[13] (11) At autopsy, was coronary occlusion with subsequent pathological changes found to correspond with the clinical history of patient and consistent with the time elapsing between claimed injury and death?

The physician must, first, set out the complete history of and the circumstances surrounding the accused event, identifying the time of onset of first symptoms; then, he should recount in detail the complete clinical findings so far as they are appropriate to the problem, including relevant personal and familial medical history.

The patient's present complaints are next in order. In fairness to the physician and to the case, the report should specifically state whether or not the conventional diagnostic aids had been resorted to. These should be identified, stating the dates and the results.

The diagnosis is, of course, the first basic conclusion the report presents. So far, we have been discussing the steps and procedures which would be a routine part of any thorough medical examination. The big difference is that in Workmen's Compensation cases, the data should be reported and identified. Then follows the identification of relationship either to the accused cause, or to some other cause discovered by the examiner. Is this an industrial injury or merely a case of illness?

There may be conflicting opinions as to causation. Not only should the doctor give the foregoing data, but, in addition, he should summarize briefly the recognized causes of the entity diagnosed, stating why he accepts the cause adopted by him and why he rejects the other possible causes. The reasons given for an opinion are most important. The weight or value of any opinion depends upon the validity of the reasons assigned for it,[14] or the facts upon which it depends.[15]

If many doctors actually reviewed in their minds the various possible causes of the condition found by them, rejecting each of them in turn for a considered reason, there would be a higher average of scientific worth in their reports.

A still more important result is obtained in that it assists the laymen who must process these reports in their problem of deciding which cause is better supported by reason. Where there are multiple possible causes and multiple examiners have reported, each adopting a different one, as only too often occurs, the laymen are left without a yardstick if the analysis suggested is not set forth.

The rules of law are clear. Difficulty arises in making the basic determination as to whether or not an injury or accident caused by the employment has occurred. The fact that the accused event "might" have caused the heart attack is not scientifically sufficient for an opinion of causation. Applying sound medical knowledge, the question is: Did it actually play a material part?

If the reporting doctor is to be swayed by "equities," let him remember that for every sympathy decision of compensability, literally hundreds, if not thousands, of cardiac cripples will be denied the chance to work.

Importance of Heart Cases in Workmen's Compensation

Vital statistics identify heart disease as the number-one killer today.[16] Furthermore, it must be apparent that more heart episodes of one kind or another occur away from work, often while asleep in bed at night, than during the regular course of work.[17]

Beard's report[18] analyzed the number of heart cases before the Commission during 1948 to 1951. The latest figures show 147,845 nonfatal injuries in California during 1957, of which 197 were cardiovascular. During 1957 there were 786 work fatalities, 156 of which, or 1 in 5, were cardiovascular. The Commission made decisions in 68. In 1958 there were 852 work fatalities, 175 of which, or approximately 20.5%, were cardiovascular. Fifty-two of these went to decision by the Commission.[20]

Of the 413 cardiovascular cases during 1957 and 1958, 211, or 67%, were based upon Compromise and Release settlements, and 102, or 33%, upon Findings and Award of the Commission. Thirty of these covered state and local law enforcement and fire protection personnel in whose favor there is a statutory rebuttable presumption of causal connection. Twenty-two covered other state and local government employees. Fifty covered employees of private firms. In 18 cardiovascular fatalities, liability was accepted by the employer or his insurance carrier.[20]

It is apparent that the number of cardiovascular deaths with some industrial connection rose to an all-time high in 1958. This resulted from an increase in the number of Compromise and Releases. Actually, fewer Findings and Awards were issued on cardiovascular death claims in 1958 than in any of the previous six years.[19] The high number of cases closed by Compromise and Release reflects the fear of the insurance companies of the hazards of litigating claimed industrial injuries involving hearts.

Opportunity Beckons

Notwithstanding the message from vital statistics, Beard's report discloses that not only are there not widely accepted hypotheses of causal connection among cardiologists, but that, being human, cardiologists are not always consistent when unknowingly reviewing the same case data.[19]

It is an unfortunate fact that as soon as litigation enters the picture, scientific procedures and attitudes become diluted by partisan enthusiasm, with the result that opinions as to the effect of an accused event upon a subsequent heart episode are produced more for their economic value than for scientific worth. Until the medical profession has clearer and more universally recognized concepts of cause and effect, it is and will remain difficult to reconcile the results desired by those seeking to rehabilitate industrially the cardiac patient and the fears of industry of an adverse effect upon compensation costs.

While a surprising unanimity of opinion was disclosed in a New York survey,[20] it is not known what percentage those reporting represented of all cardiologists practicing in New York and how many of them applied the same tests in reviewing a case with an alleged work causation.

I quote from an article prepared for a similar meeting before the Washington State Heart Association: "It is probably true that, viewed from the physician's standpoint, courts have reached inconsistent conclusions on similar or even identical factual patterns. But a careful case by case review of . . . decision will reveal that any seeming inconsistencies have resulted not so much from inconsistent application of rules of law as from variant medical opinions expressed in various cases. To one who understands the judicial process, inconsistent results may be expected where ostensibly qualified medical experts express contrary opinions regarding factual situations . . ."[21]

The respective functions performed by the legal and medical professions in manning the courts and furnishing technical expert testimony bring each group into increasing contact. It is difficult not to criticize those doctors who voluntarily enter the field of forensic or literary medicine and contribute so generously to its confusion.

There is a real need for the medical profession to formulate acceptable hypotheses of cause and effect and to recognize that every cardiac as a living human being must engage in some activity. So long as that is commensurate with his underlying health and within the range of activity to which his heart has become compensated, death should properly be assigned to natural causes unless the effect of the accused event was so substantial that any fair mind would find it responsible. So, too, should recognition be given to the time elapsing between the accused event and the heart episode. Certainly, scientific minds should question causal connection when accused cause and claimed effect are separated by days, weeks, or months.[22]

While the relation of effort to heart failure, or any of the several varieties of involvement, varies, the basic principles are the same: (1) Effort to be considered as contributory must be in excess of the daily routine; and (2) the interval between effort and the onset of symptoms or of death itself must be short, i.e., within a few hours.[23]

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Year	Total	Per 100,000
1930	25,501	446.7
1935	31,173	505.4
1940	37,593	544.2
1945	44,031	449.0
1950	53,835	508.5
1955	62,036	475.9
1957	65,879	464.2

"In 1954 and 1955 23% of all deaths in California in the age group from 35-44 were from heart disease. The proportion then went up steadily for each ten years of age—35% for the 45-54 group, 43% for the 55-64 group, and 47% for those 65 and over. . . The cardiovascular death rate for middle-aged California men is higher than in some of the other States. . ." California's Health, Vol. 14, No. 10, p. 86, Biennial Report Edition, California State Board of Public Health.

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Tetanus in Industry

The Problem with Particular Reference to Immunization Procedures

Charles D. Sherman, Jr., M.D., and David H. Barnhouse, M.D., Rochester, N.Y.

"THE MASTER of a large ship mashed the index finger of his right hand with the anchor. Seven days later, a somewhat foul discharge appeared; then trouble with his tongue —he complained he could not speak properly— his jaws became pressed together, his teeth were locked, then symptoms appeared in his neck, on the third day opisthotonos appeared with sweating. Six days after the diagnosis was made he died." Hippocrates' description of tetanus 2,300 years ago might well fit many cases throughout the world today. One hundred years after Hippocrates, Aretaeus commented, "But neither can the physician, though present and looking on, bring any assistance, as regards life, relief from pain, or from deformity." Very little was added to our knowledge of tetanus over the next 2,000 years.

At the close of the 19th century, the bacterial nature of the disease was finally discovered. In 1884, Nicolaier first caused experimental tetanus by injection of soil into animals and later described the drumstick bacilli. At the beginning of the 20th century, the science of immunology was developed and with it the possibility of immunization against certain diseases including tetanus. During the last 60 years, steady improvement in methods of immunization has been accompanied by broad programs of immunization for large groups of people.

Active immunization was made a practicality by the work of Ramon[6] in 1925 to produce tetanus toxoid. There were only five cases of tetanus in the United States Armed Services during World War II in persons who had received routine toxoid immunization and who were given a booster after injury. These occurred among nearly 3,000,000 hospital admissions for wounds. This experience contrasts very sharply with the occurrence of 2,385 cases of tetanus in the British army in World War I, of which 1,011 were fatal.

Today, in the United States, tetanus is relatively uncommon; 500 or 600 persons contract this disease each year. The importance attached to a disease of such limited incidence is justified by the following factors: It is one of the few almost completely preventable diseases which we face in this country. If a person does get the disease, his chance of dying is about 50% to 60%. Although the disease itself is uncommon, problems relating to prophylaxis face physicians even in the smallest hospitals numerous times every day. There are definite dangers in giving the antiserum; and, on the other hand, there are definite dangers in failing to give serum to wounded persons. These problems could be completely abolished by a universal program of active immunization with tetanus toxoid. If a disaster occurred affecting significant numbers of nonimmunized persons, one would expect the number of cases to skyrocket (in the 1945 battle for Manila, there were 500 cases of tetanus in nonimmunized civilians). This paper will review very briefly the general problem of tetanus followed by some comments on the problems specifically related to prophylaxis and concluding with some comments upon how these things might possibly have some significance with regard to immunization programs in industry.

Etiology

The bacterial exotoxin produced by the tetanus bacillus is one of the most powerful poisons known. One-fifth of a milligram is lethal. The disease is caused by the action of this toxin on the central nervous system. This toxin is produced by the organism which tends to remain around the local wound without invading the surrounding tissues or spreading throughout the body. Very few bacteria are needed to produce enough toxin to cause clinical tetanus, and

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Presented at the 45th Annual Meeting of the Industrial Medical Association, Rochester, N.Y., April 26, 1960.

this is one of the reasons that the organism is so difficult to isolate. The causative organism is not recovered in 30%-50% of patients who have tetanus. The organisms are strictly microaerophilic. Their normal habitat is within the gastrointestinal tract of many animals; they are not commonly found in man except when he is in contact with animals and the soil.

The spores are found in well-tilled soil that has been fertilized with animal excreta, as in Western Europe, from which area a large number of cases is reported. Such soil conditions are said not to prevail on the Western Plains of the United States. The spores are more heat-resistant than those of any other organism pathogenic to man. Boiling alone will not kill them. They may lie dormant within a healed wound only to become vegetative years later after certain changes occur in conditions about the wound. From 15% to 20% of patients with tetanus do not have a discoverable wound of entrance.

Pathogenesis

The bacilli or their spores may be carried into any wound in which either the agent making the wound or the clothing or skin of the person sustaining the wound is contaminated with Clostridium tetani. Their growth and multiplication are favored by the anaerobic conditions such as in a puncture wound or one in which there is necrotic tissue. The presence of soil minerals favors the growth of the bacteria by promoting necrosis.

The toxin produced by the bacillus acts in the central nervous system. Whether it gets to the central nervous system along the nerves or via the bloodstream is not definitely known. Once it becomes fixed in the central nervous system, it cannot be neutralized by antitoxin. To be effective, antitoxin must act on the bacterial toxin before it reaches the central nervous system. The incubation period from the time of injury to the signs of clinical tetanus may be as short as 24 hours or as long as 3 weeks. Much longer intervals are reported, but these probably represent a subsequent stimulation of dormant spores already present in the tissue rather than a true incubation period. If a person survives tetanus, there is no residual defect in any system which can be recognized as due to the bacillus or its toxin.

Clinical Features

Although occasionally the first signs of tetanus may be local muscle spasm around a recent wound, more commonly the initial symptom is trismus. If a patient who has a recent tetanus-prone wound begins to complain of tightening of his jaw, then the diagnosis of tetanus is a reasonable presumption, and treatment should be started. Treatment should not be delayed in attempts to culture or get a positive smear for the bacillus. As the disease progresses, trismus becomes more marked. Dysphagia, opisthotonos, a rigid but nontender abdomen, spasm of the facial muscles, and laryngospasm are other notable signs and symptoms.

The complications of the disease most frequently are secondary to muscle spasms and convulsions. These may be initiated by treatment procedures or inept nursing care. Trivial stimuli such as a bright illumination or jarring of a bed may trigger laryngospasm or a convulsion. Spasms affecting the larynx, the diaphragm, or the intercostal muscles may eventuate in death. The complications to be prevented or controlled include vomiting with pulmonary aspiration, pneumonia, pulmonary edema, urinary retention, cardiac problems, laryngospasm and asphyxia, drug reactions, residual spasticity of muscles, traumatic glossitis, dehydration, diarrhea, fever, fecal impaction, oversedation, and serum reactions.

Treatment

The three broad aims in therapy are (1) neutralization of toxin and prevention of any further elaboration thereof; (2) the prevention of spasms which cause acidosis through increase of lactic acid and, more importantly, respiratory paralysis; and (3) the prevention of pneumonia and other complications.

Toxin fixed by the central nervous system, of course, cannot be dealt with. Toxin which has been produced but not yet fixed is neutralized with massive doses of tetanus antitoxin (TAT). This antibody will also take care of any more toxin which may be formed at the nidus of infection. It is best to give all of the TAT initially as sensitivity and faster excretion of the foreign protein can develop if it is given intermittently. After the appropriate skin or conjunctival sensitivity tests, 50,000 immunizing units are given intravenously and another 50,000 intramuscularly. This will afford protective levels of antibody in the serum for five to six weeks. The intrathecal route is contraindicated because it not only does not afford any extra benefits but it may also damage the nervous system in its own right.

In order to prevent further production of the tetanus toxin, the local infection must be treated. This is accomplished by (1) administration of antibiotics: penicillin to control the growth of the tetanus bacilli and a broad spec-

trum antibiotic to control the symbiotic organisms often present in the wound; and (2) care of the wound itself, if one is present, through careful debridement with removal of necrotic tissue and foreign material. A grossly infected, dirty wound should be debrided and packed open. Packing with activated zinc peroxide may be of value. We do not believe that amputations should be performed simply because a wound is infected with tetanus organisms.

Control of spasms is accomplished by use of sedatives and muscle relaxants. Oversedation is to be avoided, and it is unwise to carry the use of muscle relaxants to the point where the patient cannot breathe on his own. This requires continuous artificial respiration which superimposes problems related to this factor alone. Either heavy sedation or muscle relaxants can control spasms, and numerous investigators have advocated the use of one as opposed to the other, although in the majority of instances both are used. Rectal tribromoethanol (Avertin*) is advocated but may cause severe rectal irritation. The barbiturates are probably used more commonly than other drugs for sedation, and we feel that short-acting barbiturates are the best to use. For the seriously ill patient, amobarbital is given by continuous intravenous infusion with the amount being "titrated" to the correct level of sedation. For those less seriously ill, amobarbital may be given every four to six hours intramuscularly in dosages of 0.25-0.5 Gm. in a 10% solution. The British are using chlorpromazine extensively for sedation, and its use also seems reasonable to us. Muscle relaxants such as curare, mephenesin, methocarbamol, and meprobromate should be used with caution as they may give enough respiratory paralysis to necessitate continuous artificial respiration. In more seriously ill patients with severe spasms, however, the use of amobarbital alone as a sedative to control spasms would result in serious oversedation, and these patients probably should have some combination therapy. We feel that curare is not as safe a drug as the others noted. Mephenesin is superior to curare in that it can be used without the necessity for total relaxation and controlled respirations. It can be given intravenously, by stomach tube, or by rectal tube. Cortisone does not have a significant place in the treatment of tetanus itself. It does have value in treating serum reaction when it occurs.

Tracheostomy should be done in most cases. This will prevent death by laryngospasm and give the medical team easy access to the tracheobronchial tree to help control respiratory secretions and the complications resulting from aspiration of saliva or stomach contents.

Pneumonia is one of the more serious secondary complications. It is prevented by the careful control of the secretions through the tracheostomy. Antibiotics are used prophylactically. Penicillin and the broad-spectrum antibiotics are also active against the tetanus bacillus; they are, of course, ineffective against the toxin. Cardiac arrhythmias and decompensation, fecal impaction, urinary retention, decubitus ulcers, and traumatic glossitis are among other complications preventable by excellent general care. Special attention must be paid to fluid and electrolyte balance. High humidity may prevent the inspissation of bronchial secretions, which then become difficult to remove. Oxygen may at times be indicated. Oral feedings should be avoided until the convalescent stage, and even nasogastric or gastrostomy-tube feedings will not prevent the possibility of vomiting and aspiration.

Active immunization against tetanus should be carried out during the convalescence as clinical tetanus will not render the patient immune to the disease. Should the patient not be actively immunized and should he require tetanus antitoxin for a tetanus-prone wound at a later time, the fact that he has previously received serum makes him much more likely to develop serum reaction and much less likely to receive protection from the tetanus antitoxin that he might receive.

Prognosis

Despite reports suggesting that tetanus will always have a considerable mortality rate, we believe that the prognosis varies directly with the total amount of medical and nursing care given to the patient. Except for those patients who are moribund when admitted to a hospital, we believe that almost every patient with tetanus should survive his illness if meticulous care is paid to treatment as outlined above. This type of treatment was given by Forbes and Auld,[5] who reported 100% survival in 15 consecutive patients. Because of shortage of personnel and excessive costs, this type of care is not available in all parts of the world, and in some areas mortality rates are as high as 80% to 90%. Many workers report mortality rates between 30% and 50%. The longer the incubation period, the better the outlook is in a given case. An incubation period of less than four days indicates a fulminating type of infection and is associated with high mortality rates. On the other hand, the recorded mortality

*Winthrop-Stearns, Inc., 1450 Broadway, New York, N.Y.

rates of cases with incubation periods longer than 10 days are usually less than 10%. In the fulminating type of infection, the interval between the onset of trismus and convulsions is often less than 24 hours, and this indicates a very poor prognosis; these patients rarely survive. Infants and the aged have a higher mortality than those in the intervening decades. If the wound is in the area of the head and neck, it seems that the toxin can become fixed in the central nervous system more quickly, and these patients have an increased mortality.

Immunization

Passive Immunization—Passive immunization is used in patients with wounds sustained under conditions in which there is likelihood of tetanus-spore contamination and in whom there has been no previous active tetanus immunization. Since there is a certain risk of reaction in giving horse serum, which is the usual agent containing the tetanus antitoxin, not all patients who have wounds are given tetanus antitoxin. On the obverse side of the coin, however, if a wounded patient does not receive tetanus antitoxin and subsequently develops tetanus, the physician would be considered grossly negligent. This dilemma faces industrial physicians every day, and fortunately mistakes one way or the other are very rare.

There are many other problems in connection with the use of tetanus antitoxin in passive immunization. A large number of patients who develop tetanus have injuries so small that they do not even recall having had them, and these patients obviously never get to a physician for passive immunization. Although fatal reactions to serum are rare, they do occur and are more prone to occur in people who have had previous serum therapy or in those with a positive allergic history. The incidence of delayed serum reactions including serum sickness is determined partly by the criteria used in the diagnosis. If very mild serum reactions are included, there are some reports of an incidence as high as 30% of patients given serum. Significant reactions probably occur in 5% to 10% of those patients given serum. Such reactions may be totally disabling and may even yield permanent disability. Unfortunately, negative skin tests do not always completely eliminate the possibility of an immediate reaction and, of course, have no value at all in foretelling which patients will develop delayed reactions such as serum sickness. Another problem with passive immunization is that 1,500 units is not always effective in preventing tetanus. The failures are related to massive contamination and production of large amounts of toxin in the body. Lastly, should the patient who has been given a dose of serum (whether tetanus antitoxin or other types of serum) become wounded a second time and require passive immunization, the second dose would probably be eliminated faster and be less effective.

When passive immunization is relied upon, the protection achieved is rapid but transient. One can detect antitoxin in the circulation within a few minutes. The level rises to a peak after two or three days and usually is gone in 10 days or so. Large doses of 50,000 units or more, used when one suspects massive contamination, yield protective levels for several weeks.

Active Immunization—Because of the many problems involved in passive immunization, it is recommended that programs of active immunization be developed throughout the United States.[7-9] The individual is stimulated to form his own antibodies by multiple injections of tetanus toxoid, which acts as an antigen. These antibodies are retained for much longer periods of time than are horse serum antibodies which are passively received. Moreover, the production of new antibodies can continue for a long time, and this production can be greatly increased by subsequent injections of toxoid. Toxoid injections cannot be used to achieve initial rapid immunization such as may be required by a person who has a seriously contaminated wound. The initial development of effective antibody levels (estimated at 0.1 unit per milliliter of serum) takes several weeks. Even in a person who has already been immunized against tetanus, the necessary additional rise in titer after injection of toxoid may take four to five days; and, if a person who had been previously actively immunized were to be injured in a way which might result in very rapid development of tetanus (such as severe extremity injury by a manure spreader), the injured might need protection against tetanus within the first day or two and would therefore require passive immunization as well as a toxoid booster.

The work of Evans[4] leads to a clear understanding of the optimal use of toxoid for the production of active immunity against tetanus. He found that the level of circulating tetanus antitoxin one month after a single injection of toxoid was scarcely measurable. A second dose of toxoid administered at this time stimulated a rapid rise of circulating antibody to 0.5 units per milliliter of serum, with a falloff to 0.1 unit at the end of a year. One-half of Evans' subjects were then given a third (or reinforcing)

dose of toxoid, with a resulting antitoxin titer of 10.0 units per milliliter of serum at the end of 30 days on the average. More important, however, was the fact that a level of 0.4 units per milliliter of serum was still present one and one-half years later. The other half of Evans' subjects, who were not given a reinforcing dose at one year, had a level of only 0.04 units of antitoxin per milliliter of serum at the end of one and one-half years. On the basis of this work, the two injections of toxoid one month apart are referred to as the *primary immunization* (Edsall[3]). Together with the reinforcing dose at the end of one year, the whole course is considered the *basic immunization*.

Booster doses of toxoid should be given at the time of an injury which could be associated with tetanus bacillus infection. The effect of a booster is to stimulate a rapid rise of antibody titer from the level then existing. A booster dose thus administered between the 6th and 12th month after the primary immunization has been completed may be regarded as the equivalent of the reinforcing dose normally given at the end of the first year. If a booster dose has been given within six months, the patient with a new injury need not be given another toxoid injection.

As regards the frequency of administration of routine booster doses to maintain immunization in the absence of wounds, there have been some reports of significant titers detected as long as 10 to 12 years after the original basic immunization. Even at these late dates, there is a rapid rise in antibodies from a booster shot. Because antibody titers tend to fall slowly as time passes, however, and also because the data on titers at the longer intervals are relatively scanty to date, it is probably best not to separate routine periodic boosters by more than five to six years.

Management of Injured Patients

The first question which the industrial physician is asked to answer is whether or not the patient has a wound that might possibly develop tetanus. If the wound is a tetanus-prone one, then some sort of immunization procedure should be utilized. It cannot be emphasized strongly enough, however, that, regardless of immunization history, the most important procedure in the handling of tetanus-prone wounds is adequate cleansing and debridement of the injury. As Edsall[3] states, "tetanus can occur only when susceptible tissue is infected with tetanus bacilli and no history of immunization can eliminate the necessity of removing in so far as possible the foreign material and the intruded epidermis which introduced tetanus infection and the necrotic tissue which nurtures it." Tetanus-prone wounds include (1) those more than three or four hours old; (2) definitely infected wounds; (3) wounds through skin or clothing contaminated by soil; (4) all deep and punctured wounds; (5) wounds with devitalized tissue; (6) wounds that cannot be closed properly.

Treatment of Immunized Patients—From the foregoing, it is apparent that persons who have been adequately immunized within the previous five to six years are almost certain to show reasonable response to an emergency booster dose within five days after the booster injection. In rare cases, one must consider giving tetanus antitoxin as well as a tetanus toxoid booster. This happens when (1) the injury is more than 24 hours old; (2) the last toxoid booster was more than 8 to 10 years ago; (3) there is massive contamination in a very bad injury; or (4) the patient's response is apt to be delayed or absent because of some concomitant factor such as exposure to irradiation of any sort or the use of cortisone for any reason. In these circumstances, the physician again faces the dilemma as to whether the risk of giving serum is greater than the risk of the patient's acquiring tetanus.

Treatment of Nonimmunized Patients—Patients with tetanus-prone wounds who have never had active immunization must be questioned regarding the possibility of sensitivity to horse serum. This includes questioning regarding allergic history or history of prior serum therapy of any sort. Although they are not completely reliable, we believe that intradermal or conjunctival tests or both are of value in predicting sensitivity. Patients have had immediate serious reactions even to a skin test, and these should not be done without having available at hand a tourniquet to place above the injection site, ice for application over the site of injection, and epinephrine to inject in the opposite arm. It is our feeling that the proper dose of tetanus antitoxin in the routine case seen on the day of injury is 4,000 to 5,000 units. Delay of a day or more, or presence of complications such as compound fractures, gunshot wounds, extensive tissue damage, massive infection, and the like require higher doses up to 10,000 units or more depending on the circumstances. We think it better to give all the antitoxin that will possibly be needed at the initial treatment than to give fractional doses at one- or two-week intervals.

Patients who have been given serum should

be immunized actively. It is possible to give tetanus antitoxin in one arm and tetanus toxoid in the other arm and have the toxoid effective. There is a diminution in response to the initial dose of toxoid if more than 3,000 units of antitoxin are given in the opposite arm. But because three doses of toxoid are essential in order to stimulate adequate immunization, this is insignificant.

Treatment of Serum-Sensitive Patients—If a patient is sensitive to horse serum, there are three possibilities for immunizing him in the event that there is urgent need for this. (1) Bovine serum is available in most hospitals and may be administered if after a sensitivity test the patient does not prove to be sensitive to bovine serum. (2) Desensitization can be done according to the procedure described below, following which the full dose of antitoxin may be administered with the expectation that it will be absorbed slowly. There is some evidence to suggest that it is difficult to tie up completely the patient's antibodies to horse serum and that these antibodies will react with the tetanus antitoxin so that it is not available to react with the toxin. (3) The third possibility is to use the serum of patients recently immunized against tetanus. Such serum should contain 30 to 40 units of antitoxin per milliliter. The Philadelphia Serum Exchange has some purified gamma globulin made from hyperimmune patients which is being used in a clinical experiment. This is not generally available.

Desensitization—This is only rarely needed for prophylaxis, but it should be used for treatment when indicated. The principle is to give slightly increasing doses to prevent the development of anaphylaxis. The approved procedure does not truly desensitize the patient but causes a gradual fixation of his antibodies to serum. The outline presented is from the "Handbook of Medical Treatment" by Chatton and associates.[2] Antihistaminics should be given first. Epinephrine in a syringe (1 cc of 1:1000 dilution), an icebag, and a tourniquet should be immediately available. Under constant medical observation, the following doses should be given every 30 minutes.

First dose: 0.1 cc of TAT diluted 1:10
Second dose: 0.2 cc of TAT diluted 1:10
Third dose: 0.5 cc of TAT diluted 1:10
Fourth dose: 0.1 cc of TAT undiluted
Fifth dose: 0.2 cc of TAT undiluted
Sixth dose: 0.5 cc of TAT undiluted
Seventh and subsequent doses until the administration of the entire dose: 1.0 cc undiluted TAT every 30 minutes

Should a mild reaction occur, the next lower dosage should be reused and the procedure continued as before. If the reaction is more severe, epinephrine should be given intravenously, the TAT injection site should be cooled with ice and isolated from circulation with a tourniquet, and the desensitization procedure should be either discontinued or advanced even more slowly than this schedule suggests. Cortisone and corticotrophin (ACTH), contrary to some reports, are of no value in treating immediate serum reactions; their value is in the treatment of delayed serum sickness.

Serum Reactions—It is difficult to get accurate statistics of the incidence of immediate anaphylactic shock, but this probably occurs once in every 10,000 injections of serum and is said to lead to death once in every 100,000 injections (Axnick[1]). This possibility should be avoided by taking a careful history related to allergy or previous serum therapy and by sensitivity tests. The treatment as outlined previously consists in isolating the area from the general circulation by tourniquet, applying cold to reduce absorption, and giving epinephrine in the opposite arm. Delayed reactions occur in from 5% to 30% of patients and aside from serum sickness itself include such complications as neuritis, myelitis, periarteritis nodosa, deafness, pericarditis, myocarditis, and encephalopathy.

Industry Program

We have considered in this article the problems of the patient who gets tetanus, his long hospitalization and possible death. We have further considered the problems facing the industrial physician and his dilemma as to when to give and when not to give tetanus antitoxin. Finally, we have considered the problems of the patient himself as regards serum reactions including death from the same as well as prolonged and even permanent disability. But we have not considered costs of all these things to the patient and to industry. We have made numerous attempts to ascertain the medical costs of patients who have tetanus as well as the cost in time lost and compensation paid to employees for various types of serum reactions, but this information is extremely difficult to obtain. All of these problems could be completely eliminated by a wide program of active immunization which could be done at minimal cost.

We believe that a reasonable program for immunization in industry is on a voluntary basis with a slight amount of pressure being applied outlining to the employee in lay terms the problems involved. A program along the following lines is suggested. (1) Immunize

every new employee. (2) Immunize every injured employee. (3) Immunize all persons with a history of allergy. (4) Immunize all persons who have had previous horse serum (TAT or other serum). (5) Give a booster to all employees who have had their last booster more than 5 years ago. (6) Give a booster to all employees who work or play in areas where they may get tetanus-spore contamination. (7) Reanalyze your immunization program, and see if there are other personnel who might possibly be induced to participate.

We believe that a card should be issued to each employee to carry in his wallet outlining his immunization status.* So many patients are seen in emergency rooms who don't know whether or not they have had immunization or who say they have had tetanus "shots" but don't know what kind or when they were given. These problems could be avoided by carrying a card containing this information. Other pertinent health data such as blood type, diabetes, etc., would also be a valuable addition to such a card.

Summary

1. A brief outline of tetanus as a disease is presented.

2. Problems involved in immunization procedures are discussed.

3. A program of active immunization against tetanus is strongly recommended to industrial groups.

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*Available free from Wyeth Laboratories.

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March 3 and 4, 1961

Hotel Georgia, Vancouver, B.C.

The conference is sponsored by the Greater Vancouver Health League. Frank Copithorne is chairman of the Program Committee. Featured speakers on the program will be Robert N. McMurry, Ph.D., President, The McMurry Company, consultants in industrial relations and market research, Chicago, and Jean S. Felton, M.D., Professor of Occupational Health, School of Medicine, University of California, Los Angeles. Included in the program this year will be case study workshops which will show delegates to the Conference, through the study of specific cases, the advantages of occupational health services and some of the problems which will have to be overcome if these services are to be successful in preventing illness and loss of income to working men and women. For further information, write Greater Vancouver Health League, 1300 Robson St., Vancouver 5, B.C.

Topical Review

The Toxicology of Carbon Tetrachloride

Charles E. Lewis, M.D., Sc.D., Kansas City, Kan.

CARBON TETRACHLORIDE is an excellent solvent. It is volatile, noninflammable, and relatively cheap. It is also a ubiquitous highly toxic chemical which continues to claim an occasional victim who has used it not wisely, but too well.

Of the United States production of 374,000,000 pounds in 1959, probably more than 50% has been or will be used in the manufacture of other chemicals, such as dichlorodifluoromethane (Freon 12). Much of the remainder will be used in fire extinguishers, fumigation of grain, and manufacture of rubber and as a degreasing agent or solvent. A large quantity of carbon tetrachloride will find its way into the hands of an unwary public. It will enter their homes as a constituent of several popular cleaning agents, in insecticide sprays, or upon the recommendation of a friend and by the direct purchase of "carbon-tet" from drugstores, hardware stores, and establishments selling and servicing fire extinguisher equipment.

A case of carbon tetrachloride poisoning serves to illustrate the relative importance and interaction of the three basic factors in the pathogenesis of disease—the agent, the host, and the environment. The agent, carbon tetrachloride, must be available in sufficient quantity for the host, or victim, to absorb a toxic amount. In cases of intoxication due to inhalation, the environment must permit the accumulation of toxic concentrations of carbon tetrachloride in the ambient air. This usually means a small, unventilated room. Certain factors in the host predispose to the development of pathology following exposure to carbon tetrachloride. Obesity,[30] excessive intake of alcohol,[11, 12, 13] or the presence of liver disease[13] serves to increase the susceptibility of the host. Perhaps the most important contribution of the host is the selection and misuse of a potentially dangerous material, usually because of ignorance of its toxic properties.

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Submitted for publication Sept. 29, 1960.

Absorption, Metabolism, and Excretion

Carbon tetrachloride is readily absorbed through the lungs and gastrointestinal tract. Some degree of percutaneous absorption is possible, although this is not thought to be an important factor in systemic intoxication. Because of its high degree of solubility in lipids and its relative insolubility in water, carbon tetrachloride is concentrated in the lipid phases of blood and tissues. In the dog, carbon tetrachloride is excreted unchanged primarily by the lungs.[27] In monkeys inhaling radioactively labeled carbon tetrachloride, 50% is exhaled unchanged via the lungs over a 75-day period, while the rest of the radioactivity appears in exhaled carbon dioxide and in the urine the urea and an unidentified metabolite.[19]

Despite the numerous cases of carbon tetrachloride poisoning on record, there is no reliable information on its concentration in the blood and/or its metabolism in man. This is primarily due to the difficulty in determining carbon tetrachloride in biological samples by present analytical techniques.

Mechanism of Action

When present in high concentrations, carbon tetrachloride has a narcotic effect upon the central nervous system. Its more specific toxicological effects are upon the cells of the liver and the renal tubules.

In experimental animals dosed with carbon tetrachloride (CCl_4), alterations in biochemical functions of the liver precede any evidence of microscopic damage.[25] An immediate decrease in the alkaline phosphatase of the centrallobular cells followed by a subsequent increase of this enzyme in the cells of the portal areas has been reported.[25] During the first few hours following administration of CCl_4, there is a reduction in the hepatocellular content of glutamicoxalacetic transaminase (GOT) and glutathione.[25] Increases in the concentration of xanthine oxidase and GOT in serum have been observed.[5]

Cornish and others have demonstrated that

the first day following exposure to high concentrations (6,000 ppm for four hours) a fall in the cellular concentration of esterase is accompanied by an increase in esterase in the serum. The second day after exposure, both serum and liver concentrations of esterase are depressed below normal.[6] A loss of potassium from mitochondria, with a concomitant increase in the water content of the mitochondria, occurs in liver cells treated with CCl_4.[29] Certain enzymes such as glutamic dehydrogenase are lost from the cell only at a time when fatty degeneration and necrosis of the cell become microscopically evident.[14]

These studies are consistent with the theory that carbon tetrachloride acts upon the hepatic cell to produce a progressive loss of the contents of its mitochondria. A loss of enzymes and certain important cell metabolites such as diphosphopyridine nucleotide (DPN) from the liver cell precedes uncoupling of oxidative phosphorylation or any visible alteration in the histology of the liver.[14] In support of the theory that loss of mitochondrial contents leads to cellular death, the pretreatment of rats with nicotinic acid or tryptophan (precursors of DPN) offers considerable protection against lethal doses of carbon tetrachloride.[10]

Others have proposed that the changes seen in the liver following the intake of carbon tetrachloride are due to ischemia mediated by a massive discharge of the sympathetic nervous system and its neurohumors.[3] Work by these investigators has shown that sectioning of the spinal cord prevents the changes observed in animals following the administration of carbon tetrachloride.

Recent studies have indicated that regeneration processes following the intake of carbon tetrachloride are diffuse and involve cells in intermediate areas as well as those in the central zones.[18] While necrosis and fatty degeneration are most commonly seen in the centrallobular areas, it is highly probable that all "zones" of the liver are participating in the injury and regeneration processes.

Less is known regarding the nephrotoxic actions of carbon tetrachloride. It is possible that its effects on renal tubular cells are qualitatively similar to those seen in the liver. Such effects would be amplified by the inability of the kidney to excrete carbon tetrachloride. Bodie[1] has emphasized that the renal excretion of a molecule is dependent upon its being present in filtrate in a polarized, or ionized, state. The completely un-ionized, or nonpolar, carbon tetrachloride molecule can be visualized as being filtered at the glomerulus and refluxing continuously back and forth through renal tubular cells. Renal tubular destruction due to carbon tetrachloride can be distinguished pathologically from ischemic necrosis (hemoglobinuric nephrosis), since in the former instance there is necrosis of a large segment of the proximal tubule down to, but not including, the basement membrane, while in ischemic necrosis the destruction of tubular cells is random and involves the basement membranes of the cells.[9]

Pathologic changes in severe cases of carbon tetrachloride poisoning are not limited to the liver or kidneys but may be found in almost all tissues or organs.[21]

Carbon Tetrachloride Poisoning in Man

Carbon tetrachloride poisoning may be acute or chronic. It can be due to ingestion of the liquid or inhalation of its vapors. Acute intoxication results from inhalation of vapors produced while using the material in a small, unventilated area, intentional ingestion with suicidal motives, or accidental ingestion (most common in small children and alcoholics). Chronic intoxication is usually due to industrial exposures.

Acute Intoxication Due to Inhalation—Exposure to toxic concentrations of carbon tetrachloride in the air is productive of the following symptoms: irritation of the eyes, nose, and throat; nausea; vomiting; headache, and dizziness. These symptoms frequently disappear within a few hours after cessation of exposure. Absorption of larger quantities produces stupor, convulsions, coma, and death due to depression of the central nervous system. Sudden death may occur from ventricular fibrillation or depression of the vital centers in the brain stem. After a delay of from several hours to two or three days, signs and symptoms of injury to the liver and kidneys may become evident. In some cases, initial symptoms of centrol nervous system depression are not followed by signs or symptoms of hepatic or renal damage. Conversely hepatic and renal pathology may occur in the absence of any immediate effect on the central nervous system.

Common delayed findings include nausea, vomiting, abdominal pain, diarrhea, hematemesis, rapid enlargement of the liver, tenderness in the right upper quadrant, jaundice, and abnormal liver-function tests. Wróblewski[22] states that concentrations of glutamic oxalacetic transaminase in serum reach their highest values in viral hepatitis and carbon tetrachloride intoxication. Proteinuria, hematuria, and cylindruria precede anuria.[35] Optic neuritis with decrease in the visual fields,[31] necrosis of the adrenal cortex, and pancreatitis are infrequent complications. Pulmonary edema has

been reported in some cases of carbon tetrachloride intoxication.[13] This complication is probably a result of the combination of acute renal failure and damage to the myocardium and central nervous system; it may follow either ingestion or inhalation of the solvent. An attempt has been made to implicate phosgene as a cause of pulmonary edema in some of these cases.[31] Thermal degradation of CCl_4 does result in the generation of phosgene. A study by Smyth and Smyth,[32] however, demonstrated that no phosgene was produced when concentrations of up to 5,000 ppm of carbon tetrachloride were present in the vicinity of a pilot light of a gas stove.

Exposure to 60,000-80,000 ppm for 30-60 minutes will cause either immediate or delayed death.[17] Inhalation of 15,000-30,000 ppm for longer than one hour usually produces signs and symptoms of intoxication. The highest concentration which can be breathed for brief periods of time (one-half hour) without serious consequences is 1,000 ppm.[20]

Chronic Intoxication Due to Inhalation—Continued exposure to concentrations of carbon tetrachloride in excess of 100 ppm is productive of physiological disturbances. Previous studies have shown that when carbon tetrachloride is present in levels from 100-300 ppm in the industrial environment workers may suffer from nausea, vomiting, loss of appetite, abdominal pain, apathy or mental confusion, and loss of weight.[7, 8, 33] Dermatitis has been described, but this is a consequence of the defatting effects of carbon tetrachloride on the skin.

Evidences of hepatic or renal damage by either clinical examination or laboratory tests may be present if the exposures are of sufficient magnitude and frequency.

A recent study by Kazantzis and Bomford[16] reports symptoms of nausea and vomiting in 15 out of 18 workers exposed to between 45 and 100 ppm of carbon tetrachloride in air. The threshold limit recommended by the American Congress of Governmental and Industrial Hygienists is 25 ppm. The odor of carbon tetrachloride is detectable at 72 ppm.[28] It is obvious, therefore, that the odor of carbon tetrachloride in an environment means that it is present in excess of the suggested limit for continued daily exposure.

Intoxication due to the Ingestion of Carbon Tetrachloride—Ingestion of carbon tetrachloride results in a clinical picture similar to that seen in acute intoxication due to inhalation. Gastrointestinal symptoms are severer, with increased probability of hematemesis and abdominal pain. Depending upon the amount absorbed, hepatic and renal damage may become evident within several hours or be delayed for two or three days.

The lethal dose by mouth of carbon tetrachloride is said to be as little as 2 to 4 ml for some susceptible persons.[24]

The Diagnosis of Carbon Tetrachloride Poisoning

The diagnosis of acute carbon tetrachloride poisoning is primarily dependent upon obtaining a history of the ingestion of the liquid or inhalation of its vapors. In the absence of such a history, the substantiation of this diagnosis may be extremely difficult. The fact that a patient with liver or kidney disease presents a history of exposure and possible absorption of carbon tetrachloride does not prove the diagnosis. The history of the illness and the progression of the clinical course should be compatible with that described above for carbon tetrachloride intoxication. There are no specific biochemical tests which serve to distinguish the effects of carbon tetrachloride on the liver and kidney from those of other poisons or infectious agents which exhibit similar hepatotoxic or nephrotoxic properties. The blood serum level of GOT is usually extremely high but no higher than levels seen in infectious hepatitis. The identification of the chlorinated hydrocarbon in samples of blood, urine, or alveolar air substantiates a history of exposure to the solvent. In the absence of any quantitative data in the literature, however, such information is of little use in defining the degree of exposure.

At present, the clinical differentiation of chronic liver disease produced by carbon tetrachloride is an extremely difficult problem, except in cases in which a liver biopsy demonstrates the presence of alcoholic hyalin, biliary cirrhosis, or other specific diseases. A decision as to whether repeated exposure to CCl_4 has caused permanent hepatocellular damage or aggravated preexisting liver disease must rest upon (1) a careful medical evaluation of the patient to determine the presence of other factors (hemochromatosis, biliary cirrhosis, etc.), (2) a definite history of (repeated) exposure to the agent, and (3) some information, if possible, on the range of concentrations of CCl_4 in the ambient air at the site of exposure. The latter data, while usually retrospective, may be the most important factor in formulating an opinion.

The Treatment of Carbon Tetrachloride Poisoning

The therapy of any case of poisoning should be oriented toward (1) preventing further absorption of the material, (2) increasing the rate

of the removal of the poison from the body, (3) specific treatment measures directed at preventing the poison from accomplishing its toxicological actions, and (4) supportive treatment for the patient who has suffered damage to tissues. With respect to carbon tetrachloride, it is usually impossible to prevent further absorption or enhance the rate of removal of carbon tetrachloride from the body. If carbon tetrachloride has been ingested, it should be removed by gastric intubation; and sodium or magnesium sulfate, 15-30 gm, in water should be administered as a cathartic. It has also proved difficult to alter the basic effects of carbon tetrachloride upon body cells. The primary treatment, therefore, is adept management of the clinical course of the patient.

Supportive treatment for hepatic damage includes the administration of certain agents which have been suggested in animal experimentation to be of protective value but which have not proved to be equally effective in man. These include methionine and vitamin B_{12}.[15] Calcium has been shown to have some protective and therapeutic benefit in animals and man.[23] If calcium is administered intravenously, it should be given with caution, preferably while monitoring the electrocardiogram of the patient. If there is no evidence of renal failure by the second or third day following exposure, a high-carbohydrate, high-protein diet should be instituted. Chalmers has recently reviewed the treatment of hepatic failure. The treatment of the hepatic injury must be modified by the though that in all probability acute renal failure may be forthcoming. Details of the management of anuric patients have been well outlined elsewhere.[2] The presence of concomitant renal and hepatic failure makes the clinical situation a most difficult one.

The most effective treatment of carbon tetrachloride poisoning is to prevent its occurrence. Any use of carbon tetrachloride in industry should be carefully studied and other, less toxic, solvents substituted whenever possible. When its use is necessary, strict control of the environment must be maintained, and employees should be carefully screened to exclude from exposure those with a past history of liver or renal disease. Periodic clinical and laboratory examinations should be performed on the exposed workmen.

The prevention of accidental poisoning in the home is a problem confronting all physicians. Any solution to this must include the adequate labeling of all products containing carbon tetrachloride and the education of the public concerning the proper storage and use of chemicals in the home.

Summary

1. Carbon tetrachloride is an excellent solvent, highly volatile and highly toxic. In high concentrations, it produces depression of the central nervous system. Inhalation or ingestion of toxic amounts is usually followed by evidence of injury to the liver and kidneys.

2. Hepatocellular damage is probably due to the effect of carbon tetrachloride on the mitochondria of liver cells, resulting in a progressive loss of enzymes and vital cell metabolites which leads to fatty degeneration and cellular death. Ischemia caused by swelling of the cells and vasoconstriction due to the actions of the sympathetic nervous system may contribute to the end-result. Renal tubular cells are injured (probably in similar fashion) by repeated exposure to carbon tetrachloride, which is not excreted as such in urine.

3. Treatment of carbon tetrachloride intoxication should be based on the expectation that hepatic and renal failure may occur within 48 to 72 hours following absorption of the chemical.

4. The most successful approach to treatment is prevention of carbon tetrachloride poisoning through substitution of less hazardous solvents, strict control of the working environment, and proper screening of employees working with carbon tetrachloride. Poisonings occurring in the home can be prevented only through the efforts of all physicians in the education of the public with respect to the proper use and storage of chemicals in the home.

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FIRST INTERNATIONAL CONGRESS OF AGRICULTURAL MEDICINE

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Industrial Medical Association

THE PRESIDENT'S MESSAGE

ROBERT E. ECKARDT, M.D.

ONE OF THE most important decisions made by our Board of Directors at its fall meeting in Charlotte, N.C., was to propose a by-laws change which would permit us in the future to elect our officers and directors by a mail vote of our total membership. In the past, the election of officers and directors was by a vote of the members in attendance at our annual meeting. When the IMA was a small organization, a relatively large percentage of its members attended the annual meeting. These were the interested members who knew the best qualified members of our association. Today, however, IMA membership is at, or slightly above, the 4,000 figure. About 600, or 15%, of our members are able to get to any single annual meeting. Although a large number of these are interested and enlightened members, nonetheless there is another large number of just as interested and enlightened members who, though wishing to attend the annual meeting, cannot do so for personal or other reasons. Thus the west coast, southwest, and northwest regions of our country are generally lightly represented at our meetings because of the great travel distances involved. Although our meeting this year will be on the west coast and therefore presumably well attended by west coast members, there will be many east coast and central states members who will not be able to attend. The question, therefore, is should a member or fellow who has paid his dues but is unable to attend the annual meeting be disenfranchised? Your officers and directors believe that he should not, and that the way to make sure he will not is to provide him with a mail ballot which he can mark at his leisure in his office and return to headquarters. True, of 4,000 ballots that will be sent out, perhaps only 2,000-3,000 will be returned; but even this would be a far more representative vote of our membership than the 400-600 who do attend and thus vote at our annual meeting. Undoubtedly some of our electorate will vote "blind," not knowing the full qualifications of the men for whom they vote. But has there ever been an electorate for whom such a statement is not equally applicable? With the present method of proposing candidates, it is my conviction that any nominated candidate can be voted for with assurance that he is capable.

It might be well to review our methods of selecting candidates. Candidates for the Board of Directors are proposed by a Nominating Committee made up, as required by the by-laws, of three fellows from the membership at large and three *presidents* of Component Societies. By custom, the immediate Past President usually serves as chairman of the Nominating Committee, and an effort is made to achieve the widest possible geographic representation, by committee members. The committee canvasses all officers, directors, counsellors, standing committee chairmen and Component Societies officers for suggestions of nominees. Usually more than 100 names are suggested for possible candidacy. By a process of multiple voting, the Committee then narrows the list down to 10 or 12 persons who have a clear majority of Committee votes. The names of these persons are then submitted to the Board of Directors, who usually approve but can delete, add to, or in any way they see fit modify the recommendations of the Nominating Committee, *except* that they must present *at least* two names for every opening on the Board of Directors.

A list of candidates for IMA offices is developed in a different way. The Executive Committee makes its recommendations to the Board

of Directors of nominees for President-Elect, First and Second Vice Presidents, Secretary and Treasurer. In general it is felt that such nominees should be men who have served a term on the Board of Directors, where their abilities and willingness to work have been intimately observed. Some very capable men may not be good candidates for office in IMA simply because they do not have the time that it takes to perform well. In any case the Directors have final responsibility to nominate the candidates for the offices listed above, either agreeing with the Executive Committee's recommendations or selecting nominees of their own choice. Under serious discussion at the present time by the officers and Directors is the advisability of presenting two candidates for each office, abolishing the precedent of a "line of succession" from Second Vice President to President. Conceivably some men might be superb vice presidents for 10 years, but would be unable or unwilling to serve in the top posts. It has been proposed by the Committee on Organization appointed by President Lauer last year that we add one additional Vice President to the two we already have. To do so, however, would almost require the abolition of the "line of succession," with the President-Elect selected from any one of three equal vice presidents, the Secretary, the Treasurer, or any other capable person who has at least served a term of three years on the Board of Directors. I am sure that this situation will receive much serious debate and discussion in the years to come by your officers and directors, and that their ultimate decision will be made in terms of what is best for IMA as a whole. I can assure you that all your officers and directors take very seriously the responsibility which you, the members, have placed upon them by electing them to their present positions. They wish also to be sure that they will remain responsive to your wishes, since in a democratic society, or organization, the elected officials *must* remain responsive to the will of the electorate.

Book Reviews

THE HEART IN INDUSTRY. Edited by Leon J. Warshaw, M.D. Price, $16.00. Pp. 677. Paul B. Hoeber, Inc., Medical Division of Harper & Brothers. 49 E. 33rd St., New York 16, New York, 1960.

By EDWARD M. KLINE, M.D.

This reviewer, having started to read "The Heart in Industry" with a this-must-have-all-been-said-before attitude, was surprised to find the assignment an agreeable one. In this day of competition for the precious time that remains after the day's work is done, it is refreshing to find a book for the doctor with enough depth to keep his attention and so sophisticated in its orientation to industry that it never "talks down" but only "with" the reader. Nineteen separate parameters of the subject are dealt with authoritatively by 24 contributors. Each section, though appearing in orderly sequence, bears little connection with the one that follows or precedes it; yet each is essential to the coverage of the whole. This arrangement adds to the usefulness of the text both for the casual reader and for the reader concerned at the moment with any one of the facets covered. Very little seems to have been overlooked, the completeness being illustrated by such chapter headings as "Physiologic Effect of Work," "Mass Screening," "Vocational Rehabilitation," "Psychiatric Aspects," "Cardiac Emergencies," "Peripheral Vascular Disease," "The Cardiac on the Farm," "Health Education," and many others. It is regrettable that space does not permit comment on the contribution of each author. Only a few random examples, not necessarily highlights, can be included here.

Data presented by Dr. Warshaw in the opening chapter underscore the importance of heart disease to the physician, to employee, and employer alike. It has been shown that cardiac disease accounts for 2/5 of the total mortality (between 45 and 64), and among men it is the commonest cause of hospitalization for nonsurgical conditions, the period of hospitalization being nearly twice that of the average for all conditions reported. The physician who may be called upon for, or has already rendered, expert testimony in a cardiac case contested before a workmen's compensation court will substantially increase his understanding of the problem by a careful reading of the sections dealing with this subject. He will, among other things, be better prepared to handle the dilemma created by the difficult legal questions requiring an unqualified "yes" or "no" answer. The uncertainty of this yes-or-no approach to things is not confined to the occasional witness but is shared alike by men who do this work regularly. Equally well qualified experts often disagree with each other and sometimes conflict with their own previously rendered medical opinions. Evidence is presented which indicates that in most states there is a trend toward liberalization of workmen's compensation awards for cardiac cases, both in the number allowed and in their cost. In concluding this subject, the author states:

Dr. Kline, a cardiologist, is Consulting Physician to the Lamp Division, General Electric Company, Cleveland, Ohio.

"Thus, it can be seen that although compensation awards for heart disease represent only a minute fraction of the cases allowed in most states, they involve from about one tenth to one fifth of the cost of all awards. Efforts are being directed toward the implementation of remedies for this problem . . ."

It has often been said that the threat of compensation claims in employees with heart disease is the most important deterrent to their full employment. It is reported that when the matter was studied in greater detail and depth by questionnaire and by follow-up interviews, this did not appear to be the case. The question seems to remain unanswered. Many inconsistencies and paradoxes remain.

The work physiologist is an important colleague to the industrial physician in the task of matching the man to a suitable job. Whereas precise measurements of cardiac output or caloric expenditure are too burdensome to be employed regularly, mention should be made of a simple pulse rate test discussed in the chapter "Physiologic Effect of Work on the Heart." In this procedure the worker's pulse is determined at rest, at work, and during the recovery period. From these determinations the man's fitness for a particular job can be evaluated as well as his reaction to his environment and work routine. It must be emphasized that no matter how precise the physiologic tests of work performance become, they cannot estimate the emotional components which may in some individuals overshadow the physical capacities of the organism to respond to work stress. From the chapter on "Vocational Rehabilitation of Cardiacs" some important thoughts are expressed as follows:

"When an employed person develops heart disease, there is all too often a tendency to think immediately of a drastic change of job. The outmoded idea that cardiacs can do only 'light work' or must be restricted to sedentary jobs is still, unfortunately, much too prevalent.

"This represents the fallacy that any physical activity beyond minimal levels will make a 'bad' heart worse. It often reflects the physician's inability or unwillingness to assess the functional capacities of the individual patient and his fear of condemnation should difficulties recur. The guiding principle should be not what is best (or safest) for the doctor, but what is best for the patient . . .

"Preservation of the patient's 'will to work' is the key to successful vocational rehabilitation."

Several valuable check lists accompany the section on "Cardiac Emergencies" which can be used to assess the adequacy of equipment and medication available to the occupational physician to meet these urgent situations. The table on arrhythmias (page 370) is a good one and will be of interest to dispensary personnel, both physician and nurse.

To this reviewer there has never been anything particularly magic about the term "cardiac-in-industry." He is simply the heart disease patient with a job. The difference lies between whether he is seen in his physician's private office when he is a "cardiac" or when seen by the industrial physician when he becomes a "cardiac in industry." Since both doctors are working with the same patient for the same purpose of restoring his health and employability, a book such as this should be of interest to all physicians, whether in or out of industry, who care for persons afflicted with heart disease.

THE MEDICAL DEPARTMENT OF THE UNITED STATES ARMY IN WORLD WAR II: ORTHOPEDIC SURGERY IN THE MEDITERRANEAN THEATER OF OPERATIONS. Editor in Chief Colonel John Boyd Coates, Jr., MC. Editor for Orthopedic Surgery Mather Cleveland, M.D. Price, $4.00. Pp. 368. Office of the Surgeon General, Department of the Army, Washington, D.C., 1957. For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.

THE MEDICAL DEPARTMENT OF THE UNITED STATES ARMY IN WORLD WAR II: ORTHOPEDIC SURGERY IN THE EUROPEAN THEATER OF OPERATIONS. Editor in Chief Colonel John Boyd Coates, Jr., MC. Editor for Orthopedic Surgery Mather Cleveland, M.D. Price, $4.00. Pp. 397. Office of the Surgeon General, Department of the Army, Washington, D.C., 1956. For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.

By M. LAURENS ROWE, M.D.

Largely of historical interest, the first of these volumes traces the changing policies of the Army Medical Department regarding management of extremity wounds from November, 1942, until May, 1945. During this period, while the fighting ranged 2,500 miles from Casablanca across North Africa to Bizerte, then to Sicily, and up the Italian peninsula to the Swiss border, concepts of proper care of battle wounds changed as radically as the climate.

The prewar dictums of plasma for shock, sulfonamide drugs for prevention of infection, and the closed plaster method for the management of fractures and wounds were, one by one, discarded.

Long before the end of the campaign, the necessity of whole blood to combat shock, the superiority of penicillin over the sulfonamides, and the crucial importance of adequate initial debridement of wounds had been established. Delayed primary closure of soft-tissue wounds at 5-10 days, based only upon a clean clinical appearance of the wound, was found to be possible provided original wound toilet had been adequate.

Many of the lessons of World War I had to be relearned in World War II. This volume is an attempt to record those lessons as applied to the management of musculoskeletal war trauma.

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It is a useful and interesting archive and belongs in every medical school library, but its lessons are expressed better and more briefly elsewhere, and the book has little practical value in civilian practice.

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The second book is an effort to record the lessons provided by World War II in the care of extremity wounds. It will have its chief value in the event of recurrent mass conflict. It has little clinical application to civilian medicine.

Part I outlines administrative considerations involved in providing orthopedic consultation service to the various medical units of the Army of 1945.

Part II repeats the basic lessons of the care of war wounds—adequate initial debridement and delayed primary closure. Clinical experience with individual injuries of the extremities is described and illustrated with impressive numbers of cases.

Part III is a collection of four papers on special types of bone and joint injuries and includes a review of jeep-produced fracture dislocations of the hip, battle-incurred fractures of hip joints, penetrating wounds of knee joints, and complete dislocations of acromioclavicular joints. Based upon reports made in 1946, the papers are somewhat outdated.

Like the companion volume on Orthopedic Surgery in the Mediterranean Theater of Operations, this book should be in every medical school library as a reference source. It will be of only historical or sentimental value to the average practitioner.

Questions

The questions published in this column are edited versions of requests for information that have been received during recent months. The answers have been prepared by one or more competent authorities in the particular area covered by the question. Readers may address questions to the Editor.

QUESTION: *I am now seeing in psychiatric evalution a middle aged man who has been working as a truck driver for the past twenty-five years or so. He was referred to me because of agitation and paranoid delusions. What is the likelihood that the prolonged exposure to gases in his occupation will contribute to his psychiatric condition? I shall appreciate any information you may be able to give me about this and also any references you may have.*

ANSWER: Since conditions such as "agitation and paranoid delusions" are usually the result of multiple etiological factors, to which toxic agents, if present, can contribute, it is possible that prolonged, or even acute, exposure to a gas such as carbon monoxide could contribute, along with other factors, to the psychiatric condition. In fact, when agitation is present in accompaniment of paranoid delusions one considers carefully the possibility of some toxic condition (including alcoholism), since paranoid schizophrenic patients are often not agitated whereas patients with a toxic paranoid state are. However, a paranoid person tends to try to blame his occupation for his distress, especially if there are possibilities of compensation. Hence the need for medicolegal caution in assessing such cases.

The possibility (rather than the likelihood) that such a factor as carbon monoxide could be present indicates the need for careful physical investigation for signs of intoxication, even if the mental picture is not that of an organic brain syndrome, with gross disturbances in consciousness and recent memory. Electroencephalographic study and psychological testing would be helpful to pick up evidence of brain impairment. An industrial hygiene opinion should be sought with respect to the amount of carbon monoxide exposure that could have occurred in the particular truck or trucks driven by the patient. As is true for all harmful substances, damage depends upon the intensity of the exposure and not upon the mere fact of exposure. In a motorized society, exposure to *some* carbon monoxide is a universal experience.

Suggested references are the chapter on industrial intoxications in the book "Practical Psychiatry for Industrial Physicians," by W. Donald Ross, M.D. (Charles C Thomas, Publisher, Springfield, Ill., 1956), and two sections in the three-volume "Clinical Neurology," edited by A. B. Baker (Paul R. Hoeber, New York, 1955): (1) the chapter on Cerebral Intoxication by Maynard Cohen, p. 866-943 and (2) the section by Cyril Courville on Anoxia Incident to Carbon Monoxide Poisoning, pp. 563-566.

QUESTION: *What is the relative toxicity of metallic carbonyls now in use in industry?*

ANSWER: The toxic properties of nickel carbonyl have become well known because of the numerous fatalities which have resulted in men who have been exposed to this chemical. Inhalation of nickel carbonyl is followed by headache, nausea, vomiting, and epigastric pain. After a latent period (which may range up to 24 hours) the exposed subjects may experience the onset of cyanosis, dyspnea, fever, and a sensation of substernal constriction. Cobalt carbonyl has found little use in industry. While no proper studies have appeared on this material, it would seem that its acute toxicity is less than that of nickel carbonyl. One hundred thirty milligrams per kilogram of *di*-cobalt octacarbonyl have been administered to mice without significant observed effects. While iron pentacarbonyl is evidently very toxic to rats and mice, it is oxidized spontaneously upon exposure to air. Tissue changes observed in rabbits following exposure to iron pentacarbonyl were similar to those found in rabbits exposed to nickel carbonyl. There are no reports of the consequences of exposure of men to cobalt or iron carbonyls. Skin absorption of these agents may be anticipated, however.

Editorials

Maybe Grandpa Too

THERE WAS a day not so long ago when certain aspects of scientific obstetrics and pediatrics had grandma on her ear. Her own daughter was being kept quietly in bed for 8 to 10 days to rest and give nature a chance to heal things up after a normal delivery. Granny, who had come to keep house for a fortnight, jibed us gently with recollections of how she and her friends had got out of the feather bed (or the straw tick, if it happened to be summer) to do not only the housework but the lighter field chores as well the day after giving birth to their own babes. The remonstrance was gentle, because the great satisfaction of being needed in the younger household was not to be shortened even for the sake of more rational doctoring. But her impatience with the new pediatrics was not silently borne. In her book, one fed babies when they were hungry, changed their diapers when they cried, and loved them demonstratively and incessantly; and the schedules of child care were a lot of nonsense! Today —some three schools of psychiatry, dozens of books, hundreds of new words, thousands of printed articles, and millions of grids later— it turns out that grandma was right after all.

Grandpa, not so concerned with doctoring, nevertheless also felt called upon to set the record straight once in a while. In his early years he had raised an eyebrow when a friend of his with "sugar diabetes" had been treated with a starvation diet. The patient starved to death, as gramps knew all the time he would. And then the health educators preached the necessity of emphasizing a high vegetable diet at the expense of meat in the daily food intake in order to "spare the kidneys," presumably of the work of eliminating urea and uric acid. This could be dismissed with an outspoken Bosh! and uninterrupted pursuit of the established knife-and-fork performance. But quiet resistance to the march of science became outspoken revolt when the advocates of the long life cautioned against vigorous physical exertion, particularly among the elderly in order, this time, to "spare the heart." Anyone with common sense knew that hard work never hurt anybody! The farmers with enough money to retire and move to town were the ones who died young, while those who stayed on the land and worked hard lived on and on. In this issue we present a study of atherosclerotic heart disease among lumberjacks of Finland that contributes to a growing body of evidence suggesting that maybe grandpa too knew what he was talking about.

What Is Meant by Toxic?

IN THE field of occupational medicine and industrial hygiene we have developed the bad practice of using the word *toxic* too loosely. Originally this word referred to "arrow poison," or "poison from a bow." Subsequently, ill effects of drugs were included. Still later, we spoke of toxic states, such as exist in uncontrolled diabetes, hyperthyroidism, uremia, and overwhelming infection. In all cases, a general, systemic effect was implied.

In today's literature we commonly read that a certain dust is toxic, or that a chemical which is nothing more than an irritant to the skin or upper respiratory tract is toxic. Such use is in conflict with the real meaning of the word. Additionally we find the word *toxic* applied to a chemical which under certain conditions could be toxic but which in the usual circumstances of exposure can be only a surface irritant and not a systemic poison. Formaldehyde and chlorine gas, for example, usually cause surface irritation of the eyes and upper respiratory tract without general, systemic changes. Finally, years of exposure to certain dusts may result in proliferative fibrosis of pulmonary tissue without directly causing a general, toxic state. For scientific and legal reasons this term should be used with greater care.

—Guest Editorial by
RUTHERFORD T. JOHNSTONE, M.D.

Abstracts of Current Literature

Radiation Health

The Effect of Radiation upon the Heart. M. Catterall.* J. Radiol. 387:159 (March) 1960. (*Department of Thoracic Surgery, The General Infirmary, Leeds, England.)

Clinical, radiological, and electrocardiographic examinations were performed on the hearts of 26 cancer patients before and after radiation therapy. Five males and one female had bronchial carcinoma and were treated with cobalt-60. Twenty female patients had carcinoma of the breast and were treated with 250 x-rays. There was no electrocardiographic change following the bronchial and right-chest irradiation, presumably because heart doses were low. Of these patients receiving left-chest radiation, in which the dose to the heart was in the range of 2,200 to 3,200 r, all developed ECG changes, which were confined to the T-wave. Although the time of onset was variable, the four-month and nine-month tracings were positive in every case, and the one-year tracings showed a restoration of the normal pattern. It is concluded that the effect of radiation on the heart in these cases is a temporary organic change in the myocardium. —T.S.E.

Effects of Radiations on the Heart. A. Jones* and J. Wedgwood. Brit. J. Radiol. 387:138 (March) 1960. (*Radiotherapeutic and Cardiological Departments, St. Bartholomew's Hospital, London, England.)

The question of the radiosensitivity of the heart arose early. A communication in 1897 described violent and irregular palpitation after precordial x-ray exposure. Many articles since then have described various symptomatic clinical and pathologic cardiac changes attributed to radiation, both in experimental animals and humans. Previous electrocardiographic studies have been either limited in scope or questionable in conclusion.

The present study is an electrocardiographic evaluation of 25 patients before and after therapeutic doses of x-rays involving the heart. Breast-cancer patients were used exclusively because there is usually a lack of complicating factors such as may attend intrathoracic malignancy, the patients are usually young enough to have little or minimal arteriolosclerotic involvement, and they usually live long enough to permit an extended followup evaluation. Dosimetric evaluation yielded an exposure of somewhat more than 3,000 r in 28 days to a portion of the heart, with the average cardiac dose being somewhat less. Of the 25 patients, 4 demonstrated pathologic changes in the ECG, but these were trivial and transient, and 3 of the patients had evidence of ischemic disease before radiation. Seventeen more had positional T-wave changes, and four had no demonstrable ECG changes. There were no clinical or x-ray changes. The exact mechanism of cardiac effect is still somewhat in doubt; however, previous studies and the data in this series suggest that the changes seen result from an effect of pericardial origin.

NOTE: This is a long, comprehensive article, with a good review of the literature and a good reference list. It is recommended reading for those specifically interested in cardiac effects.—T.S.E.

Excretion and Retention of Radioactive Strontium in Normal Men Following a Single Intravenous Injection. M. Bishop,* G. E. Harrison, W. H. A. Ramond, and A. Sutton. Internat. J. Radiation Biol. 2:125 (April) 1960. (*M.R.C. Radiobiological Research Unit, Harwell, Didcot, Berkshire, England.)

After a single injection of strontium-85 in two normal men, the excretion rates in feces and urine were measured up to 140 days. The body retention was followed for one year by means of a whole-body counter. The body retention, except for the first 15 or 20 days, can be fitted by a power law. Excretion of radioactive strontium appears to be a three-rate process: namely, (1) about 70% of the dose is excreted rapidly in the first 20 to 30 days primarily through the urine; (2) about 15% of the dose is excreted equally in urine and feces with a half-period of about 50 days; and (3) the remaining 15% is chronically retained with minimal excretion. These excretion-rate processes are probably linked with the degree of binding of the strontium in the lattice of apatite crystals in bone. In computing the radiation dose delivered to bone in the first 300 days after a strontium-90 exposure, nearly 80% results from the chronic retention, while another 15% results from the fraction excreted at the intermediate rate. —G.L.V.

Acute Effects of Gamma Radiation in Primates. R. G. Allen,* F. A. Brown, I. C. Logie, D. R. Rovner, S. G. Wilson, Jr., and R. W. Zellmer. Radiat. Res. 12:532 (May) 1960. (*Department of Radiobiology, School of Aviation Medicine, U.S.A.F., Randolph AFB, Texas.)

One hundred seven primates (Macaca mulatta) were exposed to 400 to 40,000 r of Co^{60} gamma rays for statistical evaluation of survival time, clinical observations, and evaluation of pathology. Exposures of more than 9,000 r resulted in severe and progressive dysfunction of the central nervous system with death resulting within three days. There was widespread injury to the gastrointestinal, respiratory, and hematopoietic organs which undoubtedly contributed to the demise of these animals. Doses of 1,500 to 10,000 r produced a marked cellular atypism in the small intestine and colon suddenly on the fifth day. Gastric and colonic ulcerations appeared, but the small intestine showed only atypical, nonfunctioning cells. The ulceration is believed to occur only when

bacterial activity is superimposed on the radiation damage. Death occurred from the fourth to the ninth day. The lower doses of 400 to 1,000 r caused a greater variability of survival times. Although failure of the bone marrow was universal, the greatest number of deaths could be attributed to uncontrolled infections and bacteremia. —G.L.V.

Surgery

Progress in the Treatment of Fractures and Dislocations, 1950-1960. T. B. Quigley* and H. Banks. New England J. Med. 263:493 (Sept. 8) 1960. (*Harvard Medical School, Boston.)

The authors present current concepts in fracture therapy in the lower extremity. Fractures of the tibia and fibula are treated simply and with greater safety when a closed reduction is done and a long leg cast applied with 40° knee flexion. War experience has demonstrated the validity of skeletal traction for tibial fractures, but the authors deplore the use of the Boehler-Braun frame, as it allows motion. Whenever possible, they prefer closed means of treatment of tibial fractures, but, when an intramedullary nail is used in a middle-third fracture, they advise a Lotte's nail. Recently in spiral tibial fractures incidental to skiing, screws and Parham bands have been used with additional casting with good results. They stress Bonnin's plea for surgical repair of major--ligament injury of the ankle, and note that with conservative therapy of the ankle there is a greater residual disability than suspected. For minor ankle-ligament injuries, they use early icing, crutches, and support with either strapping or plaster casts. Medial malleolar fractures generally should have internal fixation. In bimalleolar or trimalleolar fractures, one attempt at closed reduction is warranted, but, if unsuccessful, open reduction is preferred. Except in very stable undisplaced fractures about the ankle, a walking cast is a hazard.

Three concepts are noted in the therapy of os calcis fractures: (1) closed reduction with molding of the heel followed by a short period of immobilization and early ambulation; (2) essentially no immobilization with early activity and early weightbearing; and (3) open reduction Palmer's technique with restoration of the subtalar articular surface of the os calcis. The type of fracture will guide one in a choice of therapy.

Fractures of the talus require early accurate reduction, but in about 50% aseptic necrosis may occur. Possibly an early subtalor fusion may reduce this incidence of aseptic necrosis. In transmetatarsal fracture dislocations, accurate reduction is necessary, and injury to the dorsal pedis artery may occur. In fractures of the metatarsals, they ask for more accurate reduction, using traction if necessary, to limit residual toe stiffness and gait problems. In open fractures, the aim of therapy is to attain a closed fracture as soon as the general welfare of the patient will permit. In severe open fractures, after early cleansing and debridement, secondary closure in several days is a safer procedure than primary closure. The use of antibiotics and prophylaxis for tetanus and gas gangrene are discussed. The key points of bone grafting are noted: (1) The patient's own bone is best. (2) Calcellous bone is superior to cortical bone except when a splint is necessary. (3) When autogenous bone is impractical, bone homografts to serve as a matrix for replacement by normal autogenous bone are useful and safe. —O.E.M.

Anterior Dislocation of the Ulna at the Inferior Radio-ulnar Joint. A. P. Rose-Innes.* J. Bone & Joint Surg. 42 B, 3:515 (Aug.) 1960. (*Groote Schuur Hospital, Cape Town, South Africa.)

The clinical features and pathogenesis with two case reports of anterior dislocation of the ulna at the inferior radioulnar joint are presented. The injury is usually caused by forced extreme supination on a fixed wrist. The forearm is painfully locked in supination; the elbow moves freely, and the radiocarpal joint and hand, less rotated than the forearm, show good mobility. There is a marked furrow on the medial side of the distal extensor surface of the forearm and a slight prominence of the wrist anteriorly. Lateral displacement of the ulna gives the distal forearm a narrow appearance. Lateral x-rays show the displacement clearly, usually with a fracture of the styloid process. The authors postulate the pathogenesis as follows: (1) When the hand is fixed, torsional stresses on the forearm act on the radius through the long lever of the hand and fingers. (2) At the same time, the axis of rotation moves laterally away from the ulna. (3) Such conditions exist only when the hand is fixed so that the radiocarpal joint's independence of the radioulnar joint is lost. (4) When the force is great enough, and if the hand remains fixed, the triangular fibrocartilage is torn or detached as supination forces the distal end of the radius off the ulna. The treatment of such cases is full pronation of the forearm in the anesthetized patient with immobilization of the forearm in plaster for four to six weeks. —O.E.M.

Environmental Physiology

The Effect of Work, Clothing, and Adaptation on the Maintenance of Body Temperature in Water. W. R. Keatinge.* Medical Research Council (Great Britain), Royal Naval Personnel Research Committee Report 60/977 (Oct.) 1959. (*Department of Experimental Medicine, Cambridge, England.)

Duration of survival following accidental immersion in cold water is limited by the time required for deep body temperature to fall to critical levels (25°C or 77°F). This depends on the net rate of body heat loss, i.e., the excess of heat loss over body heat production. Factors which reduce heat loss, such as insulation by clothing or subcutaneous fat, will prolong survival.

Abstracts

There are conflicting results, however, concerning the effect of voluntary work on rate of fall in body temperature. Although voluntary work increases heat production, the increased body movement involved also increases the rate of convective heat loss by agitation of the cold water.

In the present studies, 12 naval enlisted men dressed either in permeable winter clothing or only in swimming trunks and a leather helmet were immersed in stirred water at 150°C (59°F) for 20 or 40 minutes or at 5°C (41°F) for 20 minutes. Although clothing reduced the fall in body temperature under all conditions, the rate of fall was greater in working subjects than in the same subjects sitting quietly. Immersion in cold water produced a transient decrease in end-tidal CO_2 and in a few subjects caused single or multiple ventricular extrasystoles. Previous exposures appeared to reduce the reflex shivering response to cold. The principal conclusion which these results suggest is that men accidentally immersed in cold water will survive longer if they lie quietly than if they perform voluntary work such as active swimming. —D.M.

Dermatology

Agranulocytosis and Jaundice During Therapy with Meprobamate and Promazine. J. Zweifler.* New England J. Med. 262:1229 (June 16) 1960. (*Department of Internal Medicine, University of Michigan Medical School, Ann Arbor, Mich.)

This report calls attention to three cases of major reactions to a combination of meprobamate with promazine (Prozine). The communication is of particular importance because of the high incidence of reaction in the group treated (3 of 68), the severity of the reactions (2 patients with complete agranulocytosis, 1 patient with granulocytopenia, and all 3 patients with jaundice), and the high incidence of jaundice (3 of 68) secondary to drugs that are said to rarely produce this complication. —L.J.M.

Dermatoses from Phenylmercuric Salts. G. E. Morris.* Arch. Environ. Health 1:53 (July) 1960. (*Boston, Mass.)

Morris emphasizes the role of sensitivity to phenylmercuric salts in persons developing eruptions of the legs and/or hands after spreading weed or crabgrass killers. He also draws the reader's attention to the possible role of phenylmercuric salts in causing dermatitis on other parts of the body when they are used as primary antiseptics, preservatives, and spermicidal agents in ointments and jellies. —L.J.M.

Sun Screening Substances. D. V. Stevanovic.* Brit. J. Dermat. 72:271 (July) 1960. (Department of Dermatology, University of Belgrade, Yugoslavia.)

The absorption spectra of various sunscreens are measured, and the transmission through them of the wavelengths of 290 μ-500 μ are calculated using concentrations of the chemicals customarily employed in ointments. In order of increasing transmission, the least transmission was found with esculin, followed by salol, Pyribenzamine, para-aminobenzoic acid, tannic acid, sulfanilamide, antipyrine, nicotinic acid, and resorcinol. Previous in vivo studies by other investigators cited in the paper tend to confirm the validity of the author's spectrophotometric approach. —L.J.M.

The Primary Irritant Nature of Cement. G. E. Morris.* Arch. Environ. Health 1:23 (Oct.) 1960. (*520 Commonwealth Ave., Boston, Mass.)

The major factor in the causation of cement dermatitis is probably not sensitization to chromium salts but primary irritation from the cement. Two cases are presented of primary ulcers of the skin in cement workers. —M.M.K.

An Unusual Occurrence of Sporotrichosis. H. D. Garrett* and J. B. Robbins. Arch. Dermat. 82:570 (Oct.) 1960. (*El Paso, Texas.)

Several cases of occupational sporotrichosis occurred in a group of Mexicans from injuries received while unpacking pottery. The pottery had been packed in a native grass contaminated with Sporotrichum schenckii. —M.M.K.

Allergy to Phenethicillin. L. H. Criep* and H. Friedman. New England J. Med. 263:891 (Nov. 3) 1960. (*University of Pittsburgh School of Medicine, Pittsburgh, Pa.)

An allergic, anaphylactoid reaction occurred in an atopic patient from ingestion of a tablet of phenethicillin, a new synthetic penicillin. The patient had a past history of penicillin allergy. Sensitivity to phenethicillin was confirmed by scratch tests, passive transfer, and hemagglutination tests. Cross-reaction with penicillin was demonstrated in the phenethicillin-sensitive patient, and cross-reaction with phenethicillin was demonstrated in a penicillin-sensitive patient. —M.M.K

Nylon Dermatitis. G. E. Morris.* New England J. Med. 263:30 (July 7) 1960. (*520 Commonwealth Ave., Boston, Mass.)

Allergy to nylon fabric may be due to dyes, finishes, or virgin nylon. Six cases of nylon dermatitis with positive patch tests to virgin nylon are reported. One of these cases was occupational, due to contact with a nylon telephone cord. —M.M.K.

Sensitivity to Hydrocortisone Acetate Ointment. R. Church.* Brit. J. Dermat. 72:341 (Oct.) 1960. (*The Royal Infirmary, Sheffield, England.)

Sensivity to hydrocortisone ointment, used in the treatment of eczematous conditions of the skin, is rare. Patch tests in five cases showed that the sensitizing agent was 21-diol acetate, a

precursor of hydrocortisone acetate. Sensitivity to a pure hydrocortisone salt has not yet been reported. —M.M.K.

Cutaneous Porphyria in Turkey. R. Schmid.* New England J. Med. 263:397 (Aug. 25) 1960. (*Harvard Medical School, Boston, Mass.)

About 5,000 cases of cutaneous porphyria have occurred in several Turkish provinces since 1955. The syndrome resembles porphyria cutanea tarda and consists of bullae and epidermolysis of the exposed skin, hyperpigmentation, hypertrichosis, hepatomegaly, and porphyrinuria. Epidemiologic studies incriminated hexachlorobenzene, a fungicide used in treatment of seed wheat. The wheat had been used for human consumption instead of for planting purposes. Laboratory studies showed that 0.2% hexachlorobenzene, when fed to rats, produced urinary excretion of uroporphyrin and coproporphyrin. —M.M.K.

Noise and Hearing

Influence of Loud Contralateral Stimulation on the Threshold and Perceived Loudness of Low-Frequency Tones. M. Loeb* and A. J. Riopelle. J. Acoust. Soc. America 32:602 (May) 1960. (*U. S. Army MRL, Fort Knox, Ky.)

It has been demonstrated by indirect means that the stapedius muscle in man provides as much as 20 db of effective attenuation of high-intensity sounds. The present studies were designed to measure the attenuation directly by determining the change in absolute threshold when the protective reflexes are activated. Thresholds were measured at 125, 500, and 1000 cps in one ear before, during, and after presentation of a reflex-arousing tone—2200 cps at 100 db SPL (sound-pressure level)—to the other ear. The shifts were unexpectedly small, less than 5 db. This leads the authors to suggest that the protective reflex somehow results in a greater attenuation of high-intensity than of low-intensity sounds and that it acts as an energy-limiting device rather than a resistive attenuator. This theory is supported by an experiment measuring the change in loudness of a suprathreshold 500-cps tone induced by a 2200-cps arousal tone at 105 db SPL, using loudness-matching techniques. It was found that the louder the test tone the greater the diminution of loudness produced by the arousal tone. A 70-db-SPL tone showed no change in loudness, while the loudness level of a 105-db-SPL tone was diminished by 8 db (median of 14 observers). Some speculations as to the physiological basis for an energy-limiting mechanism are offered. —W.D.W.

Relation Between Loudness and Duration of Tonal Pulses: II. Response of Normal Ears to Sounds with Noise Sensation. F. Miskolczy-Fodor.* J. Acoust. Soc. America 32:482 (April) 1960. (*Research Department, New York Eye and Ear Infirmary, 218 Second Avenue, New York 3, N. Y.)

These studies on threshold change in relation to varying durations of a tonal pulse show that the rate of threshold change is reduced and the time threshold shortened when noise is compared with a 1000 cps pure tone. The greater loudness level of low noise intensities explains this result and also indicates a direct relationship between loudness and time threshold. When the pulse duration is shortened and the frequency distribution of the test tone changes, the relationship changes. In the case of very short pulses, the rate of threshold change increases through a frequency-dependent duration range. Increased energy spread on the frequency axis may cause this. Further shortening of the tone-pulse duration results in a decreased rate of threshold change (in pure tone ranges 250-4000 cps). This rate of threshold change is similar to that for noise. The direct relationship between time threshold and loudness therefore may be altered at very brief signal durations. —H.O.P.

Relation Between Loudness and Duration of Tonal Pulses: III. Response in Cases of Abnormal Loudness Function. F. Miskolczy-Fodor.* J. Acoust. Soc. America 32:486 (April) 1960. (*Research Department, New York Eye and Ear Infirmary, 218 Second Avenue, New York 3, N. Y.)

In earlier studies, a relation between tonal duration and loudness was established for normal ears. The shortest perceptible pulse duration for a given suprathreshold level (time threshold) was found to be related directly to its loudness. This paper reports studies on this relation in ears giving an abnormal loudness response. Time thresholds were measured in some 300 cases of receptor lesions with complete loudness recruitment. Some 100 loudness balance measurements made on the same subjects and data for some 200 other balances were selected from the literature. It was found that at a constant level above threshold the hearing level is related to the logarithm of the degree of loudness increment. At a given hearing level, the degree of loudness increment decreases linearly with the logarithmic use of level above the pathologic threshold. In these cases, it appears that the loudness and the degree of loudness increment, as determined by the time-threshold test, is equivalent to that measured by loudness matching. —H.O.P.

Noise Reduction in High Pressure Fluid Transfer Systems. F. W. Vetter* and F. S. Venable. Noise Control 6:41 (Jan.-Feb.) 1960. (*Esso Standard Oil Company, Baton Rouge, La.)

Blending tetraethyllead into motor and aviation gasoline is a noisy operation. The process is carried out under vacuum, and the noise is created by pressure drops through small venturis. This process was studied to determine how the noise might be reduced. It was shown that changes in the processing system could be made—without reducing the efficiency of the process—that reduced noise levels from values some 10 db or more

above those recommended by the Subcommittee on Noise in Industry, American Academy of Ophthalmology and Otolaryngology, to values below the recommended levels. The recommended maximum levels are for the 300-600 cps band and the 600-1200 cps band 85 db re 0.0002 microbar. The authors summarize: "The studies of the cause of fluid flow noise through restrictions such as venturis reveal that unnecessarily high pressure drops are often responsible for excess noise. Simple and economical changes in pumping systems may be the only measures required to control unsatisfactory noise conditions." —H.O.P.

Clinical Toxicology

Role of the Blood-Brain Barrier in the Experimental Intoxication of Carbon Disulfide. V. Bartonicek* and C. Michalova. Folia med. 18:675, 1960. (*Institute of Industrial Medicine, Prague, Hungary.)

Previous publications established that among the various organs the brain contained the smallest amount of a given parenteral dose of carbon disulfide. Because of this observation, the authors decided to investigate the role of the so-called blood-brain barrier in its relation to carbon disulfide distribution in the mammalian organism.

Histamine and hyaluronidase were used in the attempt to modify the permeability of the blood-brain barrier. Two groups of rats, treated, respectively, with histamine or hyaluronidase subcutaneously, were given carbon disulfide by subcutaneous injection. The determinations of carbon disulfide, differentiated in two fractions, free and complexed compounds with tissue moieties, were carried out with brain and liver samples.

Rats treated with histamine presented a higher value of free carbon disulfide in the brain than the respective control group. Rats treated with hyaluronidase had a higher content of carbon disulfide in free form in the liver in comparison with the control group.

Although neither one of these differences appeared to be significant under the three sigma test, the authors devote particular attention to the histamine effect. Consequently, they emphasize the importance of an allergic condition as a factor apt to favor cerebral localization of carbon disulfide, presumably through increased circulatory levels of histamine. —M.C.B.

Cholinesterase Determination in Blood and Neurological Observations in Workers Exposed to Organic-Phosphorous Compounds. H. Bauer.* Arch. Gewerbepath. u. Gewerbehyg. 18:91 (May) 1960. (*Neurologische Universitatsklinik, Hamburg-Eppendorf, Germany.)

In the author's experience, the pseudocholinesterase activity, which is the esterase activity present in serum, constitutes the most sensitive chemical control in subjects exposed in insecticide of the Diazinon-type. The acetylcholine esterase, identified commonly as the esterase present within the erythrocytes, shows a delayed response to this exposure. Both enzyme groups may be altered before any clinical evidence of intoxication is present, but the pseudocholinesterase is lowered earlier and more significantly that the erythrocytic, or "true," esterase.

The role, the distribution, and the relative importance of these two enzyme systems in peripheral and in central nervous structures are analyzed on the basis of the author's experience and of the pertinent published data. Interesting comments are devoted to the histopathological illustrations of this toxic feature, in which edema, hyperemia, and occasional hemorrhages are the most frequent findings in nervous organs.

The exposition of the physiological chemistry underlying the intoxication, based on an extensive bibliography, makes this paper an up-to-date report on cholinesterase inhibitors of industrial occurrence.

A modified version of the Michel method is suggested for its consistency and practicability for control measurements of an exposed industrial population. —M.C.B.

The Risk of Mercurialism in Production of Thermometers. G. Jenny, A. J. Chaumont, E. Weil, and Mme. G. Jenny. Arch. mal. profess. 21:439 (July-Aug.) 1960.

This survey concerns workers exposed to inorganic mercury vapors during the production of thermometers. By a semiquantitative method, using a Drager detector, these investigators found in four different plants values of mercury which frequently exceeded the MAC of 0.1 mg/m^3, with peak values up to 0.35 mg/m^3 in some instances. The clinical examination of the 36 workers employed in these plants led to the diagnosis of seven cases of positive mercurialism and six cases of probable mercury intoxication. Erethism, as interpreted by asthenic changes, anorexia, diarrhea, and excitability were present in 41% of the exposed subjects. Tremors were detected in 36%, gingivitis in 22%, and albuminuria in 14% of this population.

The urine was examined for mercury qualitatively by the Gettler method. The urinary excretion of mercury was measured quantitatively by a method in which diphenylcarbazone was used and also by a diphenylthiocarbazone (dithizon) spectrophotometric method. A good parallelism was noticed between clinical evidence of mercurialism and the value of mercury eliminated in the urine. These authors remark, nevertheless, that in two subjects who presented the highest values of mercury excretion no sign of mercurialism was noticeable, and, for all practical purposes, they were not intoxicated.

The subjects diagnosed as sure cases of mercurialism excreted between 19 and 60 μg of mercury per liter of urine. For those considered

probable cases, the value ranged between 19 and 30 μg.

The filling of the glass capillaries appeared to be the most dangerous operation based on the air levels of mercury concentration and the frequency of clinical abnormalities encountered in the involved workers. —M.C.B.

Industrial Hygiene Controls

Myocological Contamination of Impinger Dust Samples. R. L. Kathren,* H. Blocker, and B. Tom. Am. Indust. Hyg. A. J. 21:261 (June) 1960. (*Division of Occupational Health, Los Angeles City Health Dept., Los Angeles, Calif.)

This article reports an unusual contaminant which, while not heretofore reported, may have caused confusion in some analyses of environmental dust samples. The authors noted a large number of spherical particles about 2 μ in diameter in several of their midget impinger samples which were not present in the larger Greenburg-Smith impinger samples collected from the same area simultaneously. This suggested that the midget impinger tubes were definitely contaminated, but the source of the contamination was not immediately clear. It was suggested that the contamination was either dust, bacteria, or spores from fungi. Close examination of the contaminant under high power indicated that the particles were spheres with crenated edges and of exactly the same size. This tended to rule out dust contamination. Subsequent cultures on agar, blood agar, and Sarbouraud's medium supported the hypotheses that these were mold spores. Further investigations strongly pointed to the rubber stopper used to seal the midget impinger as a source of the contaminant. They conclude: "the use of scrupulously clean glassware and propyl or isopropyl alcohol as the sampling medium should eliminate the possibility of mold contamination." —E.C.R.

Survey of Lead Hazard in Indoor Firing Ranges. C. R. Ross* and others. Am. Indust. Hyg. A. J. 21:256 (June) 1960. (*Occupational Health Division, Dept. of National Health and Welfare, Ottawa, Ontario, Canada.)

This article reports the air concentrations determined in various parts of nine firing ranges which are used intermittently for training and recreation. In spite of mechanical ventilation, which usually included both supply and exhaust most of the atmospheric samples obtained during actual firing showed lead concentrations from 10 to 100 times the allowable concentration. Fortunately, the authors were able to make urinary lead analyses which showed relatively normal values and no values in the region where lead poisoning would be expected. The increase in lead values with duration of time spent in the firing range, nevertheless, indicates a very definite potential hazard for anyone spending full time in one of these ranges. The authors conclude that the exposures should be controlled and suggest good ventilation as the solution. They recommend that a supply of tempered fresh air be distributed over the firing end and exhausted from the target end of the range maintaining a linear flow toward the targets of at least 25 ft/min. Good housekeeping to prevent contamination from redispersal of settled dust is also essential. —E.C.R.

Air Flow Calibration of Direct Reading Colorimetric as Detecting Devices. H. L. Kusnetz.* Am. Indust. Hyg. A. J. 21:340 (Aug.) 1960. (*U.S. Dept. of Health, Education and Welfare, 1014 Broadway, Cincinnati 2, Ohio.)

This article not only calls attention to a limitation of the most readily available and convenient air-sampling kits used in industrial hygiene but also presents a simple method for calibrating this type of equipment in the field. While the calibration of some of the pumps used in small portable sampling instruments was well within the plus or minus 5% which is usually considered reasonable, many of the devices were as much as 30% or more in error. This is not a predictable error. It is suggested that a simple method of checking in the field would serve to increase the reliability of these instruments.

The author's summary states: "The necessity for checking carefully the air flow characteristics of direct reading colorimetric gas detecting devices has been shown. Construction of a simple buret using a soap bubble as the indicating medium has been described. Such a device could be made part of field equipment." —E.C.R.

Developments in the Measurement of Atmospheric Sulfur Dioxide. A. F. Welch* and J. P. Perry. Am. Indust. Hyg. A. J. 21:316 (Aug.) 1960. (*Air Pollution Engineering Research, U. S. Public Health Service, Cincinnati, Ohio.)

The authors compared three methods for the measurement of sulfur dioxide in low concentrations, primarily for use in air pollution studies. They present tables showing the theoretical and measured sulfur dioxide concentrations in a stainless steel test chamber using the sodium tetrachloromercurate method (TCM) of West and Gaeke adapted to an automatic analytical instrument. The results show good correlation between theoretical mixtures and test results. In another table, the stability of sulfur dioxide samples with respect to storage time is shown, and over a period of 72 hours there is no significant change. The TCM method is also compared with the hydrogen peroxide method and a correlation equation established. A further comparison with the Thomas Autometer showed the results obtained with this instrument to be identical to those obtained with the TCM method.

Although the relationship between the TCM method and two other methods was investigated,

the authors have basically established the accuracy, sensitivity, and practicality of this method as used with an automatic analytical instrument for air pollution study. —E.C.R.

Experimental Toxicology

The Toxicity of Bromochloromethane (Methylene Chlorobromide) as Determined on Laboratory Animals. T. R. Torkelson,* F. Oyen, and V. K. Rowe. Am. Indust. Hyg. A. J. 21:275 (Aug.) 1960. (*Dow Chemical Company, Midland, Mich.)

Results of toxicity studies on bromochloromethane are as follows: the oral LD_{50} for rats is between 5-7 g/kg; the cutaneous LD_{50} for the rabbit is >5 g/kg. It is moderately irritating to the eyes and has the usual defatting action of solvents on prolonged skin contact. On single inhalation exposures, the maximum exposures causing no mortality were 0.1 hr at 40,000 ppm, 0.4 hr at 20,000 ppm, 1.5 hr at 10,000 ppm, and 7 hr at 5,000 ppm. Deaths were from anesthesia. Maximum exposures without organ changes (liver damage) were 0.025 hr at 40,000 ppm, 0.1 hr at 10,000 ppm, 0.3 hr at 5,000 ppm, 3 hr at 1,500 ppm, and 7 hr at 600 ppm. A six-month exposure, seven hr daily, of several species to 370 ppm was without significant effect. The substance has only a slight capacity for inducing liver damage. The suggested industrial hygiene standard for daily exposures is 200 ppm with 400 ppm as the absolute maximum for peak exposures. The blood bromide-ion concentration at this level is estimated at 40 mg %—well below levels which produce adverse effects. —R.E.E.

The Comparative Toxicity of 6 Organophosphorus Insecticides in the Rat. E. F. Edson* and D. N. Noakes. Toxicol. & Appl. Pharmacol. 2:523 (Sept.) 1960. (*Chesterford Park Research Station, Saffron Walden, Essex, England.)

Oral and dermal LD_{50}'s speed of recovery of blood cholinesterase activity after sub-lethal oral dose, and the effects of 1, 5, 25, or 125 ppm in the diet for 15 weeks are presented for parathion, Phenkaptone, dimeton-methyl, dimethoate, Diazinon, and Dipterex. The last five had LD_{50} values from 14-200 times higher than parathion, ranging from 44-649 mg/kg. Dermal LD_{50}'s were also higher, ranging from 443->2,800 mg/kg, compared with 75 and 100 mg/kg for the emulsifiable solution and wettable powder formulations of parathion, respectively. There was much variation in the rate of depression and regeneration of cholinesterase activity with the six compounds studied and also in relative depression of cell and plasma activity. The 100-day diet toxicity tests were negative (non-cholinesterase-inhibiting) at 1 ppm with all compounds, at 5 ppm with Phenkaptone, Diazinon, and Dipterex, and also at 25 and 125 ppm with Dipterex. The studies suggest that in recent years progress has been made in reducing mammalian toxicity while preserving insecticidal properties. —R.E.E.

Insecticide Residues in the Milk of Dairy Cows Fed Insecticides in Their Daily Ration. N. Gannon, R. P. Link, and G. C. Decker. J. Agric. Food Chem. 7:829 (Dec.) 1959. Pub. Health Eng. Abstr. 40:17 (July) 1960.

Aldrin, dieldrin, heptachlor, DDT, and methoxychlor were fed in the daily rations to dairy cows for 16 weeks. For varying periods afterward, the cows were fed on insecticide-free rations. Milk samples were analyzed through both periods to determine the rate of accumulation and decline. The rate of accumulation varied as follows: aldrin (excreted as dieldrin) > dieldrin > DDT > heptachlor > methoxychlor. —R.E.E.

A Study on the Acute Toxicity of the Tri-Aryl Phosphates Used as Plasticizers. H. F. Bondy,* E. J. Field, A. N. Worden, and J. P. W. Hughes. Brit. J. Indust. Med. 17:190 (July) 1960. (*Coalite and Chemical Products Limited, England.)

Results indicate that tri-*o*-ethylphenyl phosphate has a low order of toxicity for chickens, but the mono- and di-*o*-ethylphenyl phosphates are highly toxic. Tri-tolyl phosphate made from phenols from which all the *o*-cresol and *o*-ethylphenol are removed is low in toxicity. For tri-xylenyl phosphate, removal of all *o*-cresol and *o*-ethylphenol from the raw materials does not result in a nontoxic ester. The mono- and di-*o*-propylphenyl phosphates were found to be toxic, while the tri-substituted compound was nontoxic. The toxicity of the *o*-tolyl, *o*-ethylphenyl, and *o*-propylphenyl group is much reduced in the presence of the 3,5-dimethylphenyl group. The removal of all *o*-cresol, all *o*-ethylphenol, and all *o*-propylphenol from the raw material results in a nontoxic ester. Tri-*m*-ethylphenyl phosphate is nontoxic, but tri-*p*-ethylphenyl phosphate is highly toxic. Esters containing one or two *p*-ethylphenyl groups are nontoxic. Tri-2-methyl-4-ethylphenyl phosphate was surprisingly nontoxic. —R.E.E.

Potentiation of the Toxicity to Insects and Mice of Phosphorothionates Containing Carboxyester and Carboxyamide Groups. F. W. Seume* and R. D. O'Brien. Toxicol. & Appl. Pharmacol. 2:495 (Sept.) 1960. (*University of Wisconsin, Madison. Present address: Inst. f. Pflanzenpathologie, U. Göttingen, Germany.)

The toxicity of mice of seven phosphorothionates containing carboxyester and carboxyamide groups was potentiated by EPN (ethyl p-nitrophenyl thionobenzenephosphate) tri-*o*-cresyl phosphate, and tri-*o*-cresyl phosphorothionate in confirmation of the hypothesis that the principal mode of potentiation bu such compounds is their inhibition of carboxyesterase or carboxyamidase degradation. The toxicity of the eighth compound, however, cis-thionophosdrin, was not potentiated by EPN. The toxicity of one compound, Dowco 109, which does not contain a carboxyester or carboxyamide group, was also potentiated, but the toxi-

cities of two other such compounds were not. Potentiation was also shown in the case of the American cockroach and the housefly but to a lesser extent than in the mouse. (Authors' abstract) —R.E.E.

Medical Care Administration

Will Success Spoil the Administrator? R. E. Brown.* Mod. Hosp. 95:102 (Aug.) 1960. (*University of Chicago Clinics, Chicago, Ill.).

The "leader" school of thought endows administrators with a superiority that may subtly mislead. Success can breed a form of pride that can easily turn into delusions. "Arrogance is a natural hazard of the administrator because it is only half a step away from self-confidence." The administrator is tempted to forget that his success is due at least as much to the circumstances of his position as to his personal superiority.

A good organization can tolerate an amazing number of weaknesses. Because the administrator is absorbed in the success of the whole, he is often the last to learn the true extent of failure in segments of his organization. Everyone goofs, but some find it too difficult to admit. The code by which an organization judges its administrator demands that the blame be accepted in direct proportion to authority. —J.D.W.

Trustee's Responsibility for the Quality of Medical Care. H. R. Stephenson, Jr.* J.A.M.A. 174:149 (Sept. 17) 1960. (*516 E. North St., Greenville, S. C.)

Originally, hospitals were established for those persons who could not afford the services of a private physician and had no family or house in which they could be cared for—a sociological expression of man's concern for his fellow man. Today, hospitals have expanded their concern to embrace the entire community in all its health needs. This increase in scope has forced hospitals to adopt business-oriented policies and procedures. To assure that hospital services be paid for on some planned basis, Blue Cross was created in the 1930's and commercial insurance in the 1940's. These have been a mixed blessing. They have given financial help but have created more and more demands on hospitals and for hospitals. As more staff is needed, larger and more numerous educational programs are necessary.

Politically, the hospitals are a no man's land. They care for all persons regardless of income. For those who cannot pay, the hospitals contend there is a moral responsibility of the local government. Local governments often discount or disclaim this responsibility, and the hospital is left holding the bag. At one time, hospital trustees were selected for their philanthropy, which included the wealthy business men or industrialist and their wives or both. Today, they are picked for their business background, general knowledge, and leadership.

Hospitals today must be well-run, well-managed institutions which afford a safe environment in which medical care may be provided by competent qualified physicians. The trustee's responsibility is spelled out by either local or state law, by act of incorporation, or by its by-laws. The board must delegate, coordinate, and control activities. To administration may be delegated planning, operational finances, education, operation of physical facilities, equipment, and personnel.

The provision of medical care might well be the most important and challenging aspect of the trustee's job. The board's basic method of control of medical care is to grant to the staff the privilege of establishing its own by-laws, rules, and regulations, remembering that the use of hospital facilities in almost all hospitals is a privilege, not a right. Title, ownership, and responsibility reside in the trustees. The courts have stated that the trustees are responsible for what happens in the hospital.

The board may use the report of the Joint Commission on Accreditation of Hospitals as a basis of control. Accreditation is an outward and obvious sign to the community that proper safeguards and standards have been established to meet minimum national criteria of competence. This is a safeguard to the patient and to the trustee. If the medical staff does not by its actions fulfill and attain the standards they have established in their constitution and by-laws, the board must take whatever disciplinary action is called for. —A.B.

Sociocultural Stresses and the Physician—Patient Relationship. R. L. Buck.* J.A.M.A. 170:96 (Aug. 1) 1959. (*Department of Social Relations, Harvard University, Cambridge, Mass.)

There are many indications that a good physician-patient relationship is essential for optional patient care. A number of our social, cultural, and economic forces are acting to widen the gap between physician and patient.

The many articles in the lay press including the daily health columns create anxiety on the part of the laity about their illness, their symptoms, or about their general chance for a long and healthy life. Factors such as increased medical knowledge, awareness of disease, threat of mental illness, and the imminence of annihilation make people need their doctor more but also tend to make them more demanding and more critical. They need more reassurance and understanding.

There has been in the last generation a shift from home treatment to hospitalization. The patient no longer has the support of his family situation, and the physician often does not have the knowledge that could be gained from studying the patient in his home environment. Specialization has also done much to alter the situation, creating suspicion that the family physician may not have adequate knowledge or creating unjustifiably high expectations of treatment by a specialist.

Abstracts

Prepaid insurance may cause a tendency to treat the physician as a salaried employee or may cause the physician to view the patient as just another case record. Under the present economic stress, in order to keep up with increased living costs, physicians have had to raise their fees or handle more patients. This may affect both the patient and physician adversely. The image of the family physician as a community leader is no longer present.

The purpose of this paper is to create further awareness of the sociocultural factors which cause stress in the physician-patient relationship. Once we are aware that trouble does in fact exist, we must determine the factors involved and how they may be remedied. —A.B.

Internal Medicine

What Is Systemic Lupus? W. Dameshek.* Arch. Int. Med. 106:162 (Aug.) 1960. (*Tufts University School of Medicine, Boston, Mass.)

Speculation as to the etiology of diseases of unknown etiology remains a favorite "indoor sport" for many in our increasingly complex industrial environment. It would be helpful, indeed, if all such speculation were based on as good evidence and as competent analysis of that evidence as provided in this editorial. The author concludes that systemic lupus erythematosus is a complex autoimmune disorder with protean manifestations. This is another example of a vague "toxic" etiology gradually yielding to careful study and analysis of careful clinical and laboratory observations. There is reasonable hope that the pathogenesis of systemic lupus erythematosus may be understood sometime in the very near future. —L.W.

Diagnostic Evaluation of Oral Glucose Tolerance Tests in Nondiabetic Subjects After Various Levels of Carbohydrate Intake. H. L. C. Welkerson,* H. Hyman, M. Kaufman, A. C. McCuistion, New England J. Med. 262:1047 (May 26) 1960. (*Diabetes Field Research, Public Health Service, U.S. Department of Health, Education, and Welfare; Brighton, Mass.)

A study was undertaken by the authors to prove or disprove the concept that preparation for a glucose tolerance test by a diet of 250 to 300 gm of carbohydrate for three days is necessary. Preparation with such a diet is supposed to prevent a false positive glucose tolerance test in normal persons.

Fifty-seven nondiabetic healthy persons were studied. The results suggest that preparation for a glucose tolerance test by alteration of the diet is not necessary provided the patient's diet has been adequate to maintain normal weight; therefore, a high carbohydrate diet is not required in a person who has no history or physical evidence of restricted food intake. —S.H.S.

Sacroiliac Arthritis. Leading Article. Lancet 2: 1073 (Dec. 12) 1959.

Sacroiliac arthritis invariably occurs with ankylosing spondylitis and occurs early in this disease. The sacroiliac joints are affected in many other diseases, being associated with Reiter's syndrome in 54% of the cases of more than six years' duration. In contradistinction to ankylosing spondylitis, sacroiliac arthritis appears rather late in this syndrome. Spondylitis is not common in Reiter's disease. The peripheral joints, especially the lower limbs, are most frequently involved; also, unilateral sacroiliac involvement is more common in Reiter's disease than in ankylosing spondylitis, and periostitis of the plantar surface of the os calcis is often present.

Sacroiliac arthritis sometimes occurs in psoriasis with arthritis, ulcerative colitis, and a 10% involvement has been reported in rheumatoid arthritis. Reiter's syndrome seems to be the most likely of all the syndromes in which sacroiliac arthritis occurs. —S.H.S.

The Abnormal Appendix. J. Jewers.* Lancet 2: 1169 (Dec. 26) 1959. (*Weymouth and District Hospital, England.)

It has been estimated that 100 people die each month of appendicitis in the British Isles; physicians should therefore be constantly on guard and be acquainted with variations in symptoms and location of the appendix.

1. *High retrocecal*—The pain is epigastric, or right-sided; pulse rate and temperature remain normal until the disease is well advanced. Tenderness may be absent. Vomiting is not a common feature. Tenderness should be searched for by compressing the cecum from in front and behind the abdomen. Red blood cells may be found in the urine, and care must be taken not to diagnose "renal colic."

2. *Retromesenteric or retroileal*—There is sudden onset of symptoms, with diarrhea and vomiting. The diarrhea is a direct pointer to the diagnosis. An area of tenderness just above the medial to McBurney's point is highly suggestive.

3. *Pelvic*—The pain is central or hypogastric. There is diarrhea, some vomiting, and not uncommonly frequency of urination. Tenderness is slight, over the pubis, above the right inguinal ligament, and even in the *left* iliac fossa. The diagnosis can be made early by rectal examination.

4. *Obstructed*—There is more severe pain, more frequent vomiting, greater tenderness and muscle guarding, and a higher pulse rate and temperature than in the acute catarrhal inflammation.

The above report is the author's own experience in a series of 1,238 appendectomies; 11% were high retrocecal, 5% were retromesenteric, and 9% were pelvic. —S.H.S.

About the Authors

► MARTTI J. KARVONEN, M.D., Ph.D., is Director of the Physiological Department, Institute of Occupational Health, Helsinki, Finland. Originally from Viipuri (which now belongs to USSR), he studied medicine at the University of Helsinki, where he took his M.B. degree in 1940 and M.D. in 1945. During the years 1945-47 he worked as a British Council Scholar at the University of Cambridge, England, and in 1950 took the degree of Ph.D. at that University. Since 1951 he has been a lecturer (docent) of physiology with the Medical Faculty of the University of Helsinki.

Dr. Karvonen's research interests have centered round the physiology of sport and work, and fetal physiology. He started his career in the Helsinki University Institute of Physiology, being a member of the staff 1945-1950. In the latter year, he switched over to his own department in the newly established Institute of Occupational Health in Helsinki. In addition to this position, he taught applied physiology at the Helsinki University School of Physical Education during the years 1949-1956, being the Director of the Institute, 1953-1956. Since 1956, he has served in the Finnish Air Force as its Chief Medical Officer. He is the author of numerous articles in his field of physiology.

Dr. Karvonen has paid three visits to this country. The most recent one was during the 1960 Winter Olympics at Squaw Valley. He was physician to the Finnish team, having previously organized the physiology pretesting of the long distance champion skiers.

Dr. Karvonen has actively directed two field studies on cardiovascular disease in Finland, the first in 1956 and the latter in 1959, both carried out as integral parts of an international undertaking centered in this country at the University of Minnesota. The 1959 study is the basis of the article in this issue of which Dr. Karvonen is senior author, "Cardiovascular Studies on Lumberjacks."

Associated with Dr. Karvonen in this publication are his fellow workers in the research project:

P. M. RAUTAHARJU, M.D., currently a research associate in the Biophysics Departments, University of Minnesota, Minneapolis, Minn. He is a native of Tuusmemi, Finland, and received the M.D. degree from the University of Helsinki in 1959.

ESKO J. ORMA, M.D., an internist with a background in physiology. His special field of research has been gerontology. He has published several important studies on gerontological and geriatric subjects as well as on experimental atherosclerosis. He took his M.B. degree at the University of Helsinki in 1948 and the M.D. degree in 1952. He started his career in the University's Institute of Physiology, where he spent the years 1952-1957. Except for a year spent as a research associate in this country at the Laboratory of Physiological Hygiene, University of Minnesota, Dr. Orma has since 1957 held the appointment of Physician, Helsinki University Medical Clinic.

SVEN M. PUNSAR, M.D., a cardiologist, physician at the Wihuri Research Institute and the Helsinki University Medical Clinics since 1954. He took his M.B. at Helsinki University in 1950 and the M.D. degree in 1953. In 1957-1958 he spent a year as a research fellow at the Laboratory of Physiological Hygiene of the University of Minnesota. He has published both experimental and clinical studies. Dr. Punsar has carried on epidemiological studies of cardiovascular disease in Italy, the U.S., and Finland.

JUHA TAKKUNEN, M.D., who took the M.B. degree in 1954, and the M.D. in 1959. Since graduation, he has been engaged in cardiovascular research, partly in Wihuri Research Institute, Helsinki, and partly as a member of the epidemiological team whose work is reported in the present issue.

► DAVID M. SPAIN, M.D., is the author of "Occupational Physical Exertion and Coronary Atherosclerotic Heart Disease," in this issue. He is a graduate of the University of Maryland School of Medicine, Baltimore. He was formerly Director of Laboratories and Research and Medical Examiner of Westchester County, New York. He is now Clinical Professor of Pathology, State University of New York, College of Medicine at New York City, Brooklyn, N.Y., and Director of Pathology of Beth-El Hospital, Brooklyn. He is a member of the American Association of Pathologists and Bacteriologists and of the College of American Pathologists.

► JAMES C. PATERSON, B.Sc. (Med.), M.D., O.B.E., is author of the article in this issue, "Stress, Intimal Hemorrhage, and Coronary Occlusion." His first publication on this general subject, "Vascularization and Hemorrhage of the Intima of Arteriosclerotic Coronary Arteries" (*Arch. Path.* 22:474, 1938) was the original description of intimal hemorrhage in coronary thrombosis. This has been followed by some 30 more published contributions to the subject, in addition to articles dealing more generally with cardiovascular disease.

Dr. Paterson graduated in medicine from the University of Toronto (Canada) in 1925, following which he had three years of graduate training in pathology at the same university. He was a member, in 1938-1940 of the original Medical Advisory Committee to the National Research Council of Canada. He has been President of the American Society for the Study of Atherosclerosis and Chairman of the World Health Organization Study Group on the Classification of Atherosclerotic Lesions. He is now a member of the American Heart Association's Committee on Strain and Stress on the Heart and of the Committee on Lesions, of AHA's Council on Arteriosclerosis. He received the O.B.E. for medical investigation work during World War II. He retired from military service with the rank of lieutenant colonel. Dr. Paterson is Professor of Medical Research at the Univer-

About the Authors

sity of Western Ontario and Pathologist to Westminster Hospital, Department of Veterans' Affairs, London, Ontario.

► HAROLD F. MCNIECE is Professor of Law and Dean, St. John's University School of Law, New York, N.Y. His article in this issue, "Problems of Cardiac Disability and Death Claims," is based on a preliminary report of a study, begun in 1958 and directed by him, on "The Legal Basis for Awards in Cardiac Cases."

Dean McNiece has his LL.B. degree from St. John's University, *summa cum laude;* and his J.S.D. degree from New York University, New York. After a year in practice he became an instructor in law at St. John's in 1946. He moved upward in faculty rank to Professor of Law and Assistant Dean in 1954, became Associate Dean in 1957, and was appointed Dean in 1960. He is admitted to practice before the New York State and federal courts, including the U.S. Supreme Court. Since 1956 he has been a Special Hearing Officer for the U.S. Department of Justice.

Dean McNiece has over 20 published articles in legal periodicals and is the author of two textbooks. He participates in the work of several committees concerned with community and legal affairs, being currently Vice Chairman, Workmen's Compensation and Employer's Liability Insurance Law Committee of the American Bar Association. In 1955 he received the Distinguished Service Award of the Brooklyn (N.Y.) Junior Chamber of Commerce. He is a member of the American, New York State, and Brooklyn Bar Associations.

► CHARLES D. SHERMAN JR., M.D., is senior author of the article in this issue, "Tetanus in Industry." He is Assistant Professor of Surgery at the University of Rochester (N.Y.) School of Medicine and Dentistry, a diplomate of the American Board of Surgery, and a Fellow of the American College of Surgeons.

Dr. Sherman received the B.S. degree from the University of Florida, Gainesville, in 1942, and the M.D. degree from the Johns Hopkins University, Baltimore, Md., in 1945. After an internship in surgery at Duke Hospital, Durham, N.C., he continued in surgery training at the U.S. Naval Hospital, Oakland, Calif., and at the U.S. Veterans' Administration Hospital, Wadsworth, Kan.

Dr. Sherman went to the University of Rochester as a postgraduate fellow in physiology and pathology in 1948, and then became an assistant resident in surgery. In 1950 he went to Memorial Hospital, New York, N.Y., as a fellow in surgery for a year, and returned to Rochester. He was a trainee of the National Cancer Institute during the years 1949-1953. He has published extensively on cancer surgery, particularly with respect to reconstruction of the esophagus.

DAVID H. BARNHOUSE, M.D., coauthor of "Tetanus in Industry," received his M.D. degree from Columbia University College of Physicians and Surgeons in 1954. He is a Resident in Surgery at the University of Rochester School of Medicine and Dentistry. —EDITOR

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People and Events

► MARCUS B. BOND, M.D., assumed the position of Headquarters Medical Director of the Western Electric Company on January 1, according to an announcement by E. J. SCHOWALTER, M.D., General Medical Director for the Company.

DR. BOND will head a newly organized department which, when fully staffed next spring, will consist of four physicians, five nurses, two technicians and administrative personnel. The expanded medical staff will serve about 6,500 Western Electric employees in the New York City area. Medical facilities will be located in the Company's new 31-story office building at 222 Broadway, New York City, which is scheduled for occupancy early this year.

DR. BOND graduated in 1946 from Westminister College, Fulton, Mo., with a B.A. degree. He received his M.D. degree in 1949 from Washington University, St. Louis, Mo. He enlisted in the U.S. Army in 1950 and served as medical officer in the 25th Infantry Division during the Korean War. He was awarded a Bronze Star in 1952 for his service in that conflict. From 1952 to 1957, he engaged in private practice in Kansas City, Mo.

In July, 1959, DR. BOND joined the Bell System, after completing

Marcus B. Bond, M.D.

two years of study as a Fellow in industrial medicine at Kettering Laboratory, University of Cincinnati. He then served for one year as the first in-plant resident physician in occupational medicine for the American Telephone & Telegraph Company. He was awarded a doctorate in industrial medicine from the University of Cincinnati in 1960.

He is a member of Phi Beta Pi medical fraternity, the American Medical Association and the Industrial Medical Association.

► ROBERT L. RALEIGH, M.D., became Plant Physician at the Texas Eastman Company, Longview, Texas, on January 1. His appointment was announced by DAVID C. HULL, Vice-President and Manager of Texas Eastman Company, and JAMES H. STERNER, M.D., Medical Director of Eastman Kodak Company.

DR. RALEIGH has been with Eastman Kodak since 1956 as a member of the medical staff at Kodak Park Works in Rochester, N.Y. Prior to joining Kodak, he served as physician for the Student Health Service at Union College, Schenectady, N.Y. He succeeds CHARLES E. LEWIS, M.D., who recently resigned to become Associate Professor of Preventive Medicine at the University of Kansas School of Medicine.

DR. RALEIGH received a B.A. degree from Denison University, Granville, Ohio, and his M.D. degree from Albany Medical School, Albany, N.Y., in 1953. He served in the U.S. Marine Corps for two years beginning in 1944. He is a member of the Industrial Medical Association.

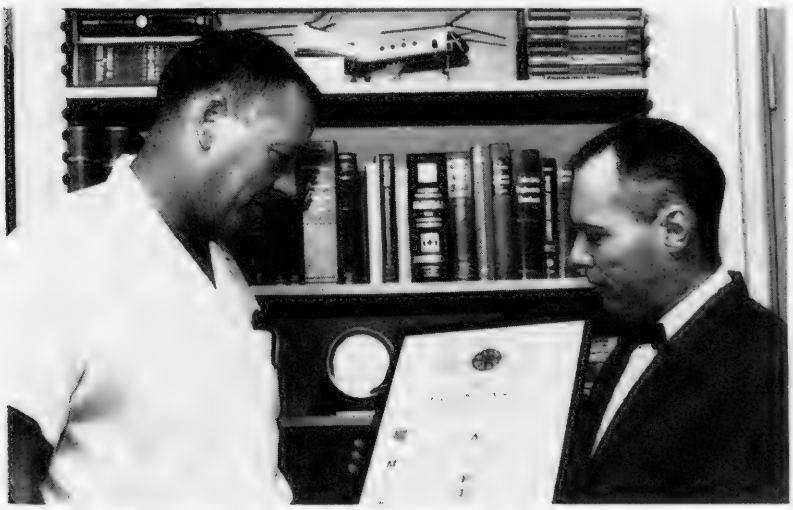
► A combined meeting of the Central States Society of Industrial Medicine and Surgery and the Minnesota Academy of Occupational

Pictured at the combined meeting of the Central States Society of Industrial Medicine and Surgery and the Minnesota Academy of Occupational Medicine and Surgery are (left to right) Gerald Gordon, M.D., John Williams, M.D., Rev. Meredith Hogue, John S. Hirschboeck, M.D., Paul J. Whitaker, M.D., Eugene J. Nordby, Mrs. R. M. Watrous, Donald Birmingham, M.D., Charles T. Disney, M.D., and Merrill P. Benoit, M.D.

Speakers at the combined meeting of the Central States Society of Industrial Medicine and Surgery and the Minnesota Academy of Occupational Medicine and Surgery were (left to right) Gerald Gordon, M.D., Richard P. Jahn, M.D., Donald J. Birmingham, M.D., Charles T. Disney, M.D., and John S. Hirschboeck, M.D.

Medicine and Surgery was held Saturday, December 3, at the Milwaukee Inn, Milwaukee, Wisconsin. There were 150 registrants for the meeting. The program was developed under the chairmanship of M. P. BENOIT, M.D., AC Spark Plug Division, General Motors Corp., Milwaukee.

PAUL J. WHITAKER, M.D., President, Central States Society of Industrial Medicine and Surgery, presided at the morning session. Addresses of Welcome were given by N. W. BOURNE, M.D., President of Medical Society of Milwaukee County, and E. J. NORDBY, M.D., Speaker of the House of Delegates, Wisconsin State Medical Society. The morning program included two presentations, one by R. P. JAHN, M.D., on "Pulmonary Problems in Industry," and the other, entitled "Radiation and Man," by C. T. DISNEY, M.D. DR. JAHN is Medical Director of the Wisconsin Anti-Tuberculosis Association and Assistant Clinical Professor of Medicine, Marquette University School of Medicine. DR. DISNEY is Medical Director, General Motors Technical Center, Warren, Michigan.



The Medical Department of Pan American's Guided Missiles Range Division at Patrick Air Force Base and Cape Canaveral has been awarded the Occupational Health Institute's Certificate of Health Maintenance. Pan American's Medical Director L. P. La Roche, M.D., right, is shown with Vice-President R. S. Mitchell.

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People and Events

A talk entitled "Current Trends in Medical Education" was given at the luncheon by J. S. HIRSCHBOECK, M.D., Dean of Marquette University School of Medicine. DR. HIRSCHBOECK stressed the growing emphasis on research in medical schools, often at the expense of preparing students for the practice of medicine. He listed several suggestions to assist medical schools—including shortening basic medical courses and making them less expensive. He also urged physicians to encourage talented young persons to consider careers in medicine.

At the afternoon session R. W. GOLTZ, M.D., Minneapolis, President of the Minnesota Academy of Occupational Medicine, presided. DONALD J. BIRMINGHAM, M.D. of the U.S. Public Health Service, Cincinnati, gave a presentation entitled "Dermatologic Problems Associated with the Manufacture of Newer Resinous Materials." GERALD GORDON, M.D., Chief of the Psychiatric Service, E. I. duPont de Nemours and Company, Wilmington, Del., spoke on "The Man and His Job."

A program for the ladies consisted of a morning meeting and a tour of the War Memorial Art Museum on Saturday afternoon.

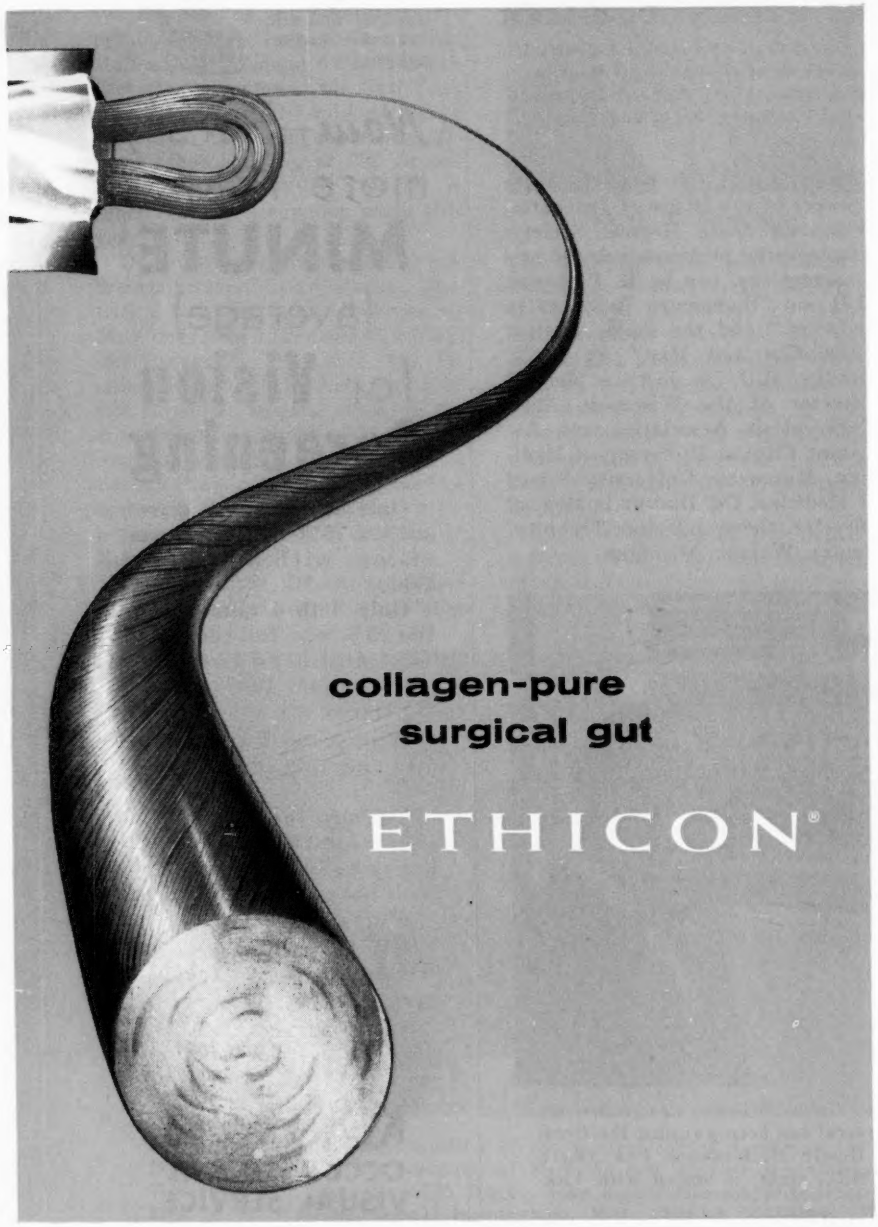
A breakfast meeting of the Board of Governors of the Central States Society was held on Sunday, December 4. ROBERT E. ECKARDT, M.D., President of the Industrial Medical Association, and CLARK D. BRIDGES, IMA Managing Director were guests.

► HARRY E. DICKSON, M.D., has been appointed Assistant Plant Physician for the Missile Systems Division, Raytheon Manufacturing Company, Andover, Mass. In this capacity, DR. DICKSON will report to WILLIAM G. SIMONIS, M.D., who is responsible for all medical functions in the Andover plant.

DR. DICKSON graduated from the University of Tennessee School of Medicine, receiving his medical degree in 1951. He interned at Baptist Memorial Hospital, Memphis, Tenn., and later received his M.S. degree from Harvard University School of Public Health, majoring in industrial hygiene and occupational health.

For the last three years, DR. DICKSON has been Director, Occupational Health Service, and Physician-in-Charge, Georgia State Employees Health Service. Prior to that he was Senior Industrial Physician with the Union Carbide Nuclear Company, Oak Ridge, Tenn. He served with the U.S. Navy during 1944 and 1945. He is a member of the American Medical Association the Industrial Medical Association and the American Association of Governmental Industrial Hygienists.

► Newly elected officers of the Section on Industrial Medicine and Surgery of the Southern Medical Association are as follows: Chairman—A. N. (SAM) HOUSTON, M.D., New Orleans; Chairman-Elect—JAMES L. HUGHES, Greer, S.C.; Vice-Chairman—JAMES FRENKIL,



M.D., Baltimore, Md.; Secretary—WILLIAM L. MACON, M.D., St. Louis, Mo. These men will be responsible for preparing the Section's program for the Southern Medical Association's 1961 meeting to be held in Dallas, Tex., November 6-9.

► The Council on Occupational Health of the American Medical Association has announced that the 1961 Congress on Industrial Health will be held at the Brown Palace Hotel in Denver, Colo., October 2, 3, and 4. The preliminary program will soon be announced.

► JOHN A. HAGAN has been appointed a Rehabilitation Consultant for the American Heart Association, according to an announcement by GEORGE E. WAKERLIN, M.D., Medical Director of the Association. MR. HAGAN was formerly State Supervisor for the Pennsylvania Bureau of Rehabilitation. In his new position as assistant to FREDERICK A. WHITEHOUSE, M.D., Director of Rehabilitation, he will help plan and develop education, research and demonstration programs conducted by Heart Associations throughout the country. These programs, which aid communities in the rehabilitation and employment of cardiovascular patients, include such activities as Work Evaluation Units to aid heart patients in returning to work, and projects which benefit young people, homemakers, farmers, and aging persons with cardiovascular disease.

► Canada's 17th National Health will be held March 12 to 18. It is sponsored by the Health League of Canada in cooperation with the Departments of Health and of Education. The Health League, a Citizen's Committee of the World Health Organization, is vitally concerned with conserving and improving standards of individual and public health. In an effort to close the gap between scientific knowledge and the application of that knowledge to daily living, the Health League seeks to bring the public into full partnership with the official agencies, through community organization and education, so that the services offered by the various Departments of Health may be fully utilized. Every form of publicity

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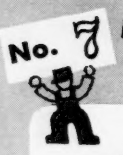

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They asked us how to combat hard-to-remove soils!

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will be used during National Health Week to further the cause of health education and action.

► New York State Society of Industrial Medicine, Inc., held an interim election at a recent meeting at which the following new officers and members of the Executive Committee were elected: Vice-President—GERALD J. FRIEDMAN, M.D.; Secretary-Treasurer—RAYMOND J. MURRAY, M.D.; Executive Committee members—CHARLES P. GIEL, M.D.; FRED W. HOLCOMB, M.D.; NORMAN PLUMMER, M.D.; and IRVING TABERSHAW, M.D.

► Industrial Medical Association of New Jersey will hold its March meeting at the Ford Assembly Plant at Mahwah, N.J., on March 17. A tour of the plant will be followed by a dinner and a business and scientific meeting.

► Industrial Medical Association of Philadelphia met January 25 at the Penn Sherwood Hotel. The program featured a discussion on "Pitfalls in Diagnosis of Post Traumatic Neuroses" by DAVID J. LAFIA, M.D., of the Neuro-Surgery Department, Jefferson Medical College. DR. LAFIA is also Director of the LaFia Neuro-Clinic of Philadelphia. The next meeting of the Association will be February 15.

► Western New York Society of Industrial Medicine and Surgery will hold its winter meeting on February 25 at the Manger Hotel; Rochester, N.Y.

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